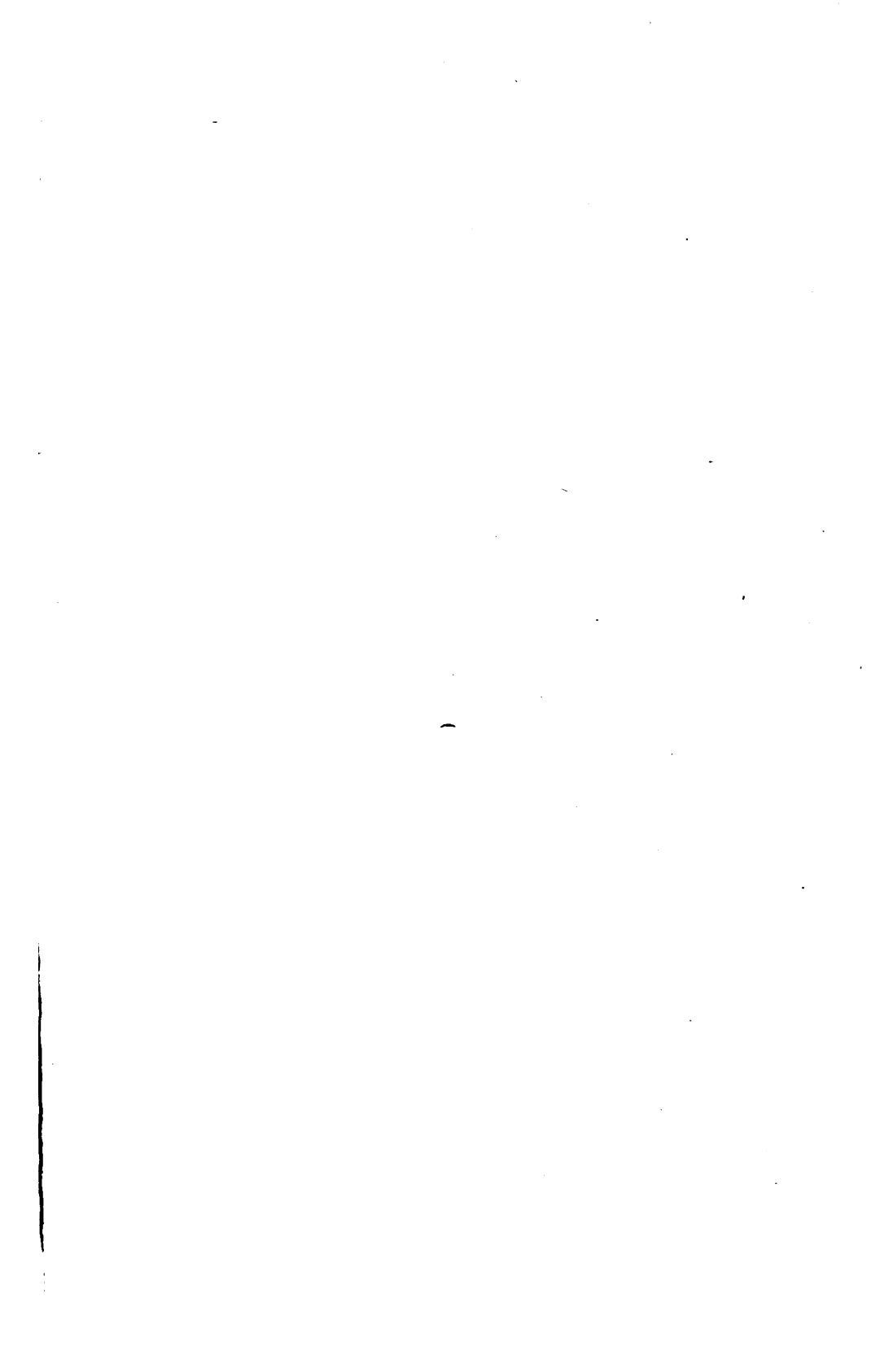


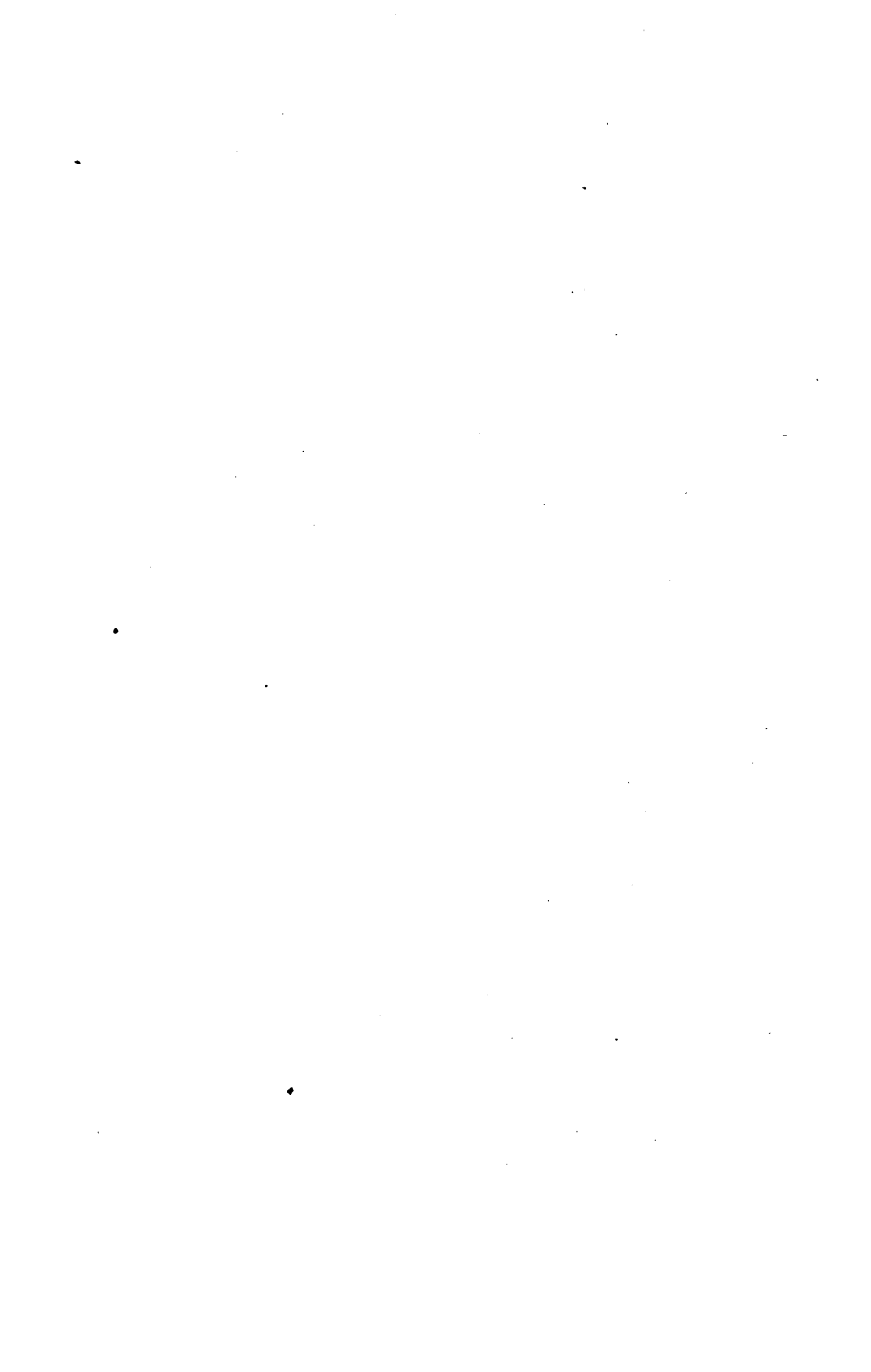


EXCHANGE











# GUY'S HOSPITAL REPORTS.

EDITED BY

F. J. STEWARD, M.S.,

AND

HERBERT FRENCH, M.D.

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VOL. LXVI.,

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VOL. LI. OF THE THIRD SERIES.



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## CONTENTS.

---

	PAGE
I. In Memoriam: Frederick William Pavy, M.D., F.R.S., F.R.C.P. By Frederick Taylor, M.D.	1
II. Cases of Poisoning by Opium. By Frederick Taylor, M.D. ... ..	25
III. Notes from the Throat Department. By F. J. Steward and W. M. Mollison ... ..	51
IV. Reports from the Aural Department:	
1. On the Results of Hearing Tests on Twenty- five Normal Individuals. By W. M. Mollison	65
2. Three Cases of Head Injury with Deafness. By W. M. Mollison ... ..	72
3. The Results of Operation Performed for the Relief of Chronic Suppurative Otitis Media. By V. Glendining ... ..	76
V. Observations on the Temperature of Man after Traumatic Section of the Spinal Cord. By H. Gardiner, M.B., B.S., and M. S. Pembrey, M.A., M.D. ... ..	87
VI. Neurological Studies (Second Series). Edited by Arthur F. Hertz, M.A., M.D. Oxon., F.R.C.P.	109
VII. Hereditary Familial Congenital Hæmorrhagic Nephritis. By George Kendall, F.R.C.P., M.R.C.S., and Arthur F. Hertz, M.A., M.D., F.R.C.P. ... ..	137
VIII. Congenital Intestinal Occlusion. By N. I. Spriggs, M.D., B.S. Lond., F.R.C.S. Eng. ... ..	143

	PAGE
IX. Gall-Stones. Colic Due to Blood Clot. By R. P. Rowlands ... ..	219
X. Metabolism During Rectal Feeding. By N. Mutch, M.A., M.B., B.C., and J. H. Ryffel, M.A., B.C., B.Sc. ... ..	223
XI. The External Abdominal Ring and its Importance in the Radical Cure of Inguinal Hernia. By Philip Turner, M.S. ... ..	239
XII. Two Examples of Opalescent Serous Fluids. By J. H. Ryffel and E. M. Mahon ... ..	251
XIII. The Results of Operation for Glaucoma. By Alan H. Todd, M.B., B.S., B.Sc. ... ..	257
XIV. The Ætiology of Melena Neonatorum, with Special Reference to Duodenal Ulcer. By Wilson Tyson, M.A., M.D. Cantab. ... ..	303
XV. A Case of Traumatic Aneurysm of the Heart. By Herbert French ... ..	349
XVI. Ante-Mortem Thrombus in Left Auricle Producing a Ball-and-Socket Type of Mitral Stenosis with Presystolic Bruit. By Herbert French	353
XVII. Ante-Mortem Clot in the Right Ventricle. By Herbert French ... ..	357
XVIII. Reduction of Blood Pressure by Iodide of Potassium. By Herbert French ... ..	361
XIX. An Unusual Type of Melanotic Growth in the Liver. By Herbert French ... ..	365
XX. A Case of Adrenal Hypernephroma in a Young Girl. By Herbert French ... ..	369



	PAGE
XXI. Calcareous Concretions Expecterated in the Sputum in a Case of Phthisis in a Young Man. By Herbert French ... ..	375
XXII. Carcinoma of the Left Kidney. By Herbert French ... ..	379
XXIII. A Simple Method of Letting Blood by Means of a Short, Wide, Hollow Needle. By Herbert French ... ..	389
List of Gentlemen Educated at Guy's Hospital who have passed the Examinations of the several Universities or obtained other Distinctions during the year 1911 ... ..	393
Medallists and Prizemen for 1912 ... ..	399
The Physical Society for 1911—1912 ... ..	401
Clinical Appointments held during the year 1911 ...	401
Dental Appointments held during the year 1911 ...	406
Medical and Surgical Staff, 1912 ... ..	408
Medical School Staff—Lecturers and Demonstrators	410
The Staff of the Dental School, 1912... ..	413

# LIST OF ILLUSTRATIONS.

## PLATES.

	TO FACE PAGE
Portrait of Frederick William Pavy, M.D., F.R.S., F.R.C.P. ... ..	1
Dr. N. I. SPRIGGS.	
Illustrating his Paper on Congenital Intestinal Occlu- sion ... ..	150, 158
Dr. WILSON TYSON.	
Illustrating his Paper on The <i>Ætiology of Mekena</i> Neonatorum ... ..	333, 334
Dr. HERBERT FRENCH.	
Illustrating his Paper on a Case of Traumatic Aneur- ysm of the Heart ... ..	349
Dr. HERBERT FRENCH.	
Illustrating his Paper on Ante-mortem Thrombus in Left Auricle Producing a Ball-and-Socket Type of Mitral Stenosis, with Presystolic Bruit... ..	355
Dr. HERBERT FRENCH.	
Illustrating his Paper on Ante-mortem Clot in the Right Ventricle ... ..	359
Dr. HERBERT FRENCH.	
Illustrating his Paper on a Case of Adrenal Hyper- nephroma in a Young Girl ... ..	370, 373
Dr. HERBERT FRENCH.	
Illustrating his Paper on Calcareous Concretions Ex- pectorated in the Sputum in a Case of Phthisis in a Young Man ... ..	378



	TO FACE PAGE
Dr. HERBERT FRENCH.	
Illustrating his Paper on Carcinoma of the Left Kidney ... ..	386

WOODCUTS, DIAGRAMS, AND CHARTS.

Mr. H. GARDINER and Dr. M. S. PEMBREY.	
Illustrating their Paper on Observations on the Tem- perature of Man after Traumatic Section of the Spinal Cord ... ..	105, 106, 107
Dr. ARTHUR F. HERTZ.	
Illustrating his Paper on Neurological Studies	123, 124, 126
Dr. N. I. SPRIGGS.	
Illustrating his Paper on Congenital Intestinal Oclu- sion	145, 147, 151, 152, 157, 160, 166, 167, 181, 189
Mr. PHILIP TURNER.	
Illustrating his Paper on The External Abdominal Ring, and its Importance in the Radical Cure of Inguinal Hernia ... ..	243
Mr. ALAN H. TODD.	
Illustrating his Paper on The Results of Operation for Glaucoma ...	275, 276, 282, 283, 284, 287, 288, 289, 290, 292, 294, 295, 298, 299, 300
Dr. WILSON TYSON.	
Illustrating his Paper on The Ætiology of Melæna Neonatorum, with Special Reference to Duodenal Ulcer ... ..	321, 330

	PAGE
Dr. HERBERT FRENCH.	
Illustrating his Paper on Reduction of Blood Pressure by Iodide of Potassium   ...   ...   ...   ...	362
Dr. HERBERT FRENCH.	
Illustrating his Paper on a Case of Adrenal Hyper- nephroma in a Young Girl...   ...   ...   ...	372
Dr. HERBERT FRENCH.	
Illustrating his Paper on Carcinoma of the Left Kidney   ...   ...   ...   ...   ...   ...	382
Dr. HERBERT FRENCH.	
Illustrating his Paper on A Simple Method of Let- ting Blood by Means of a Short, Wide, Hollow Needle   ...   ...   ...   ...   ...   ...	390, 391

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Eloum, Lieutenant-Colonel, D., M.D., 92, Gloucester Street, Warwick Square, S.W.  
Elkington, G. W., Guy's Hospital.  
Elliott, C. C., M.D., B.S., Sea Point, Cape Town, South Africa.  
Elliot, H. H., Guy's Hospital.  
Ellis, G. G., 61, Sandgate Road, Folkestone.  
Elphinstone, R., Caerleon, Ravensbourne Road, Bromley, Kent.  
Emms, A. Wilson, J.P., M.D., Belgrave, Leicester.  
English, D. C., M.D., Post Office Box 87, New Brunswick, New Jersey, U. S. America.  
Ensor, C. A., Tisbury, Salisbury.  
Erulkar, A. S., Guy's Hospital.  
Esilman, A., Guy's Hospital.

Evans, G., M.B., Heath Asylum, Bexley, Kent.

Evans, H. W., Guy's Hospital.

Evans, J. R., Dale House, Llandilo, Carmarthen.

Every-Clayton, L. E. V., M.D., B.S., Rosslyn, Clevedon,  
Somerset.

Ewart, J. H., Eastney, Devonshire Place, Eastbourne.

Eyre, J. W. H., M.D., M.S., Guy's Hospital.

Fagge, C. H., M.S., 3, Devonshire Place, W.

Fagge, R. H., High Street, Melton Mowbray.

Fasken, N. E., Guy's Hospital.

Fawcett, J., M.D., B.S., 66, Wimpole Street, W.

Fawcus, R. A., Guy's Hospital.

Fawsitt, Thomas, 46, Union Street West, Oldham.

Ferguson, G. L., Guy's Hospital.

Fisher, W. H., M.A., M.B., B.C., Oak Street, Fakenham,  
Norfolk.

Fison, A. H., D.Sc., 147, Dartmouth Road, Cricklewood, N.W.

Fitzgerald, G. H., Guy's Hospital.

Fleury, Major C. M., R.A.M.C., c/o Messrs. Holt & Co., 3,  
Whitehall Place, S.W.

Forbes, J. T. T., Guy's Hospital.

Forman, E. Baxter, J.P., M.D., Newmount, Chalfont St. Giles,  
Bucks.

Forrest, A. G., Guy's Hospital.

Forsyth, D., M.D., D.Sc., 74, Wimpole Street, W.

Fortescue-Brickdale, J. M., M.A., M.D., B.Ch., 52, Pembroke  
Road, Clifton, Bristol.

Forty, D. H., Edbrook, Wotton-under-Edge, Gloucestershire.

Foster, C. M., M.D., 34, Roxborough Street, Toronto, Canada.

Fox, H. E. Croker, M.B., Charlbury Lodge, near Weymouth.

Fox, H. J., 38, Prince of Wales Road, Norwich.

Fraser, J. A., Western Lodge, Romford, Essex.

French, Herbert S., M.A., M.D., B.Ch., 62, Wimpole Street, W.

Fremantle, F. E., M.A., M.B., M.Ch., Zetland House, Maze  
Pond.

Fripp, Sir Alfred, K.C.V.O., C.B., M.S., 19, Portland Place, W.

Fry, A. Cradock, B.A., M.B., Oxford House, Wokingham,  
Berks.

Fuller, Courtenay J., 66, Plumstead Common Road, Woolwich.

Fuller, F. H., Scandia, Birchwood Road, Parkstone.

Galabin, A. L., M.A., M.D., Tapley, Bishopsteignton, Teign-  
mouth.

Galbraith, D. H. A., Guy's Hospital.

- Galloway, W. D., Guy's Hospital.  
Gardiner, J. N., B.A., M.D., B.C., Dunmow, Essex.  
Garner, W. L., B.A., M.B., B.C., The Limes, Ampthill, Beds.  
Garrard, J. L., Guy's Hospital.  
Garrard, N., Guy's Hospital.  
Garrett, G. W. B., Guy's Hospital.  
Genge-Andrews, G. E., Guy's Hospital.  
Gibb, C. de W., Guy's Hospital.  
Gibson, C. C. G., Guy's Hospital.  
Gibson, F. G., M.A., M.D., Papanui Road, Christchurch, New Zealand.  
Gibson, J. H., The White House, Lansdown Road, Aldershot.  
Gilford, H., 205, King's Road, Reading.  
Gill, J. Macdonald, M.D., "Wyoming," Macquarie Street, Sydney, New South Wales.  
Gill, L., Guy's Hospital.  
Gillingham, A., 439, High Road, Chiswick, W.  
Glanville, L. S. H., M.D., North Street, Emsworth, Hants.  
Glenn, C. H., B.A., M.B., B.C., Muster Green, Hayward's Heath.  
Glover, C., Guy's Hospital.  
Glover, E. N., Guy's Hospital.  
Godding, H. C., Guy's Hospital.  
Godson, A. H., B.A., M.B., B.C., 63, Union Street West, Oldham.  
Godson, F. A., B.A., M.B., B.C., Pinehurst, Barlow Moor Road, Didsbury, Manchester.  
Godson, J. H., B.A., M.B., B.C., Linden House, Cheadle, Cheshire.  
Golding-Bird, C. H., B.A., M.B., Pitfield Cottage, Meopham, Kent.  
Goodhart, G. W., M.A., M.D., B.C., Guy's Hospital.  
Goodhart, Sir J. F., Bart., M.D., 25, Portland Place, W.  
Gosse, H. W., Eccleshall, Staffordshire.  
Gould, C. H., Guy's Hospital.  
Graham, L. A. J., Guy's Hospital.  
Granger, E. B., Little Milton, Tetsworth, Oxon.  
Gray, H. M., Guy's Hospital.  
Greaves, E. H., The College, Tirril, Penrith.  
Greaves, H. N., M.D., George Town, British Guiana.  
Green, A., M.B., Newbold Gate, Chesterfield, Derbyshire.  
Green, A. W., 4, Wardrobe Place, St. Paul's Churchyard, E.C.  
Green, C. L., Guy's Hospital.  
Greene, C. W., B.A., M.B., M.C., F.R.C.S., The Esplanade, Rochester.  
Greenway, C. M., 291, High Street, Plumstead, Woolwich.  
Greenwood, E. Climson, 1, Hanover House, Regent's Park, N.W.



- Grice, J. W. H., Guy's Hospital.  
 Gripper, G. D., Guy's Hospital.  
 Grove, W. Reginald, B.A., M.D., B.C., St. Ives, Hunts.  
 Groves, H. S., Guy's Hospital.  
 Growse, W., B.A., Dudley House, Kenilworth.  
 Gruggen, W., 67, Durban Road, Watford.  
 Guinness, A. F. G., Guy's Hospital.  
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- Habershon, S. H., M.D., 88, Harley Street, Cavendish Sq., W.  
 Haine, C. F., Guy's Hospital.  
 Hall, F. W., M.D., M.S., 18, College Street, Hyde Park,  
 Sydney, New South Wales.  
 Hall, Staff-Surgeon R. W. B., R.N., H.M.S. *Active*, Home Fleet  
 Halstead, D. V., Guy's Hospital.  
 Halstead, O., Guy's Hospital.  
 Hammond, J. A. B., M.B., Carlyon, Hope Road, Shanklin, Isle  
 of Wight.  
 Hampton, F. A., Guy's Hospital.  
 Hanafy, M. Z., Guy's Hospital.  
 Hancock, A. C., Guy's Hospital.  
 Hanreck, S., Guy's Hospital.  
 Hare, Lieut.-Colonel E. C., I.M.S., c/o King, Hamilton & Co.,  
 Calcutta.  
 Harkness, A. H., Guy's Hospital.  
 Harper, C. H. L., Guy's Hospital.  
 Harries, T. D., Grosvenor House, Aberystwith.  
 Harrington, F. J., Guy's Hospital.  
 Harris, E. B., St. Dunstan's, Muswell Hill, N.  
 Harris, R., M.B., 7, Queen's Road, Southport.  
 Harrison, S. S. B., Guy's Hospital.  
 Harsant, J. G., M.D., The Hive, Exeter Road, Bournemouth.  
 Harsant, W. H., Tower House, Clifton Down Road, Clifton,  
 Bristol.  
 Harvey, J. S. S., M.D., 1, Astwood Road, Cromwell Road,  
 South Kensington, S.W.  
 Haynes, C. G., Guy's Hospital.  
 Hearn, R. J., Guy's Hospital.  
 Hearnden, W. R. G., Guy's Hospital.  
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 Heatherley, F., M.B., B.S., Endellion, Rock Ferry, Birkenhead,  
 Cheshire.  
 Heckels, G. W., Guy's Hospital.  
 Helsham, C. T., Guy's Hospital.

- Henderson, H. J., M.B., B.S., The Red House, Amersham,  
Bucks.
- Henderson, T. E., Guy's Hospital.
- Henry, A. M., Guy's Hospital.
- Henry, M. G., Guy's Hospital.
- Hertz, A. F., M.A., M.D., B.Ch., 1, Weymouth Street, W.
- Hetley, Henry, M.D., Beaufort House, Church Road, Norwood,  
S.E.
- Hewetson, Major H., R.A.M.C., Easthams, Hendon Lane,  
Finchley, N.
- Hickman, H. V., M.B., The Hollies, Wanstead, N.E.
- Hicks, H. T., 56, Friar Gate, Derby.
- Hicks, R. G., 126, High Street, Ramsgate.
- Higgins, C., 52, Brook Street, W.
- Hilbers, H., 49, Montpelier Road, Brighton.
- Hills, A. Phillips, Carlton House, Prince of Wales Road, Batter-  
sea Park, S.W.
- Hinchliff, C. J., Southbrook, 1, Princess Road, S. Norwood, S.E.
- Hind, Wheelton, M.D., Roxeth House, Stoke-on-Trent.
- Hirsch, V. R., Guy's Hospital.
- Hoby, H. J., Guy's Hospital.
- Hogarth, B. W., M.D., B.S., 11, Erving Terrace, Morecambe.
- Hogben, H. F. T., Guy's Hospital.
- Holden, G. Walter, M.D., The Agnes Memorial Sanatorium,  
corner Sixth and Hyde Park Avenues, Denver, Colorado,  
U.S.A.
- Hole, Surgeon K. H., R.N., M.B., c/o Admiralty, Whitehall,  
S.W.
- Holman, F. K., M.D., Galeston, Eton Avenue, South Hamp-  
stead, N.W.
- Holman, H. J., 1, Hardwick Road, Eastbourne.
- Holmes, R. A., Guy's Hospital.
- Holmes, T. E., M.A., M.D., B.C., Portland Lodge, Knighton,  
Leicester.
- Hood, Donald W. C., C.V.O., M.D., 43, Green Street, Park  
Lane, W.
- Hopkins, C. L., B.A., M.B., B.C., City Asylum, York.
- Hopson, M. Fred., Guy's Hospital.
- Hopson, Montagu F., 64, Harley Street, W.
- Horsley, H., 81, London Road, Croydon.
- Horsley, L., Guy's Hospital.
- Howard, J. A., Clifton House, Church Road, Upper Norwood,  
S.E.
- Howard, R., M.A., M.D., B.Ch., Devon House, Buckhurst Hill,  
Essex
- Howard, Wilfred, New Buckenham, Norfolk.
- Howe, G. H., Guy's Hospital.

- Howe, J. D., 94, Stephenson Terrace, Deepdale Road, Preston.  
 Howell, J., M.B., B.S., 7, Imperial Square, Cheltenham.  
 Howell, J. B., 35, Earlsfield Road, Wandsworth Common, S.W.  
 Howell, T. A. I., 59, Upper Richmond Road, Putney, S.W.  
 Howitt, H. Sterne, Guy's Hospital.  
 Howse, Sir Henry, M.S., The Tower House, Cudham, Sevenoaks.  
 Hudson, A. B., 907, Government Street, Victoria, B.C., Canada.  
 Hughes, E. C., M.A., M.B., M.C., 26, St. Thomas's Street, S.E.  
 Hugill, George F., M.D., 197, High Road, Balham, S.W.  
 Hugo, P. J., Guy's Hospital.  
 Hull Medical Society, c/o R. C. Annandale, Queen Street (Book-seller and Stationer), Hull.  
 Hunôt, F. C., Guy's Hospital.  
 Huntley, E., M.B., B.S., Friar Street, Sudbury, Suffolk.  
 Hutchinson, F. E., Echuca, Victoria.  
 Hyde, W. S., Guy's Hospital.
- Ingram, P. C. P., M.B., B.S., Ivington, 23, Stow Park Avenue, Newport, Mon.  
 Iredell, C. E., M.D., 1, Weymouth Street, Portland Place, W.  
 Isaacs, B., Guy's Hospital.
- Jackson, P. J., 56, Tierney Road, Streatham Hill, S.W.  
 Jackson, T. L., B.A., M.B., B.C., Holly House, Cheadle, Cheshire.  
 Jacobson, W. H. A., M.A., M.Ch., Lordine Court, Ewhurst, Hawkhurst, Sussex.  
 Jalland, W. H., D.L., J.P., St. Leonard's House, York.  
 James, H. G., Guy's Hospital.  
 Janmahomed, H. I., M.D., B.S., F.R.C.S., The Bower, Pedder Road, Bombay.  
 Jaynes, V. A., 157, Jamaica Road, S.E.  
 Jephcott, C., M.A., M.B., B.C., 12, Upper Northgate Street, Chester.  
 Jimenez, R., San José, Costa Rica, Central America.  
 John, D. W., Guy's Hospital.  
 Johnson, E. E., Guy's Hospital.  
 Johnson, S. E., Guy's Hospital.  
 Jones, B., M.D., 33, Florence Road, Boscombe, Bournemouth.  
 Jones, D. R., Guy's Hospital.  
 Jones, E. Lawrence, Guy's Hospital.  
 Jones, G. B. H., Guy's Hospital.  
 Jones, G. H. West, Southgate, Eckington, Derbyshire.  
 Jones, J. Gaymer, Guy's Hospital.  
 Jones, J. M. Phillips, Guy's Hospital.

- Jones, J. P., Guy's Hospital.  
Jones, Robert, 11, Nelson Street, Liverpool.  
Jones, R. O. H., Guy's Hospital.  
Jones, W. Makeig, M.D., Beaumont, Torquay.
- Keates, Captain H. C., M.D., B.S., I.M.S., Dera Ghazi Khan,  
Punjab, India.  
Keer, K. J. T., Guy's Hospital.  
Kelsey, Fleet Surgeon A. E., R.N., Parkgate, Reigate, Surrey.  
Kemp, G. L., M.D., Worksop, Notts.  
Kemp, J. W., Guy's Hospital.  
Kendall, G., Battle, Sussex.  
Kendall, N. E., Guy's Hospital.  
Kennaway, E. L., D.M., B.Ch., Guy's Hospital.  
Ker, Hugh Richard, Tintern, 2, Balham Hill, S.W.  
Kidd, W. A., M.D., B.S., 12, Montpelier Row, Blackheath, S.E.  
Kidman, G. E., Guy's Hospital.  
Kilroe, W., Guy's Hospital.  
King, G. W., Guy's Hospital.  
Knaggs, R. Lawford, M.A., M.D., M.C., 27, Park Square, Leeds.  
Knott, F. A., Guy's Hospital.  
Kyle, J., Guy's Hospital.  
Kynaston, A. E. F., Poplar and Stepney Sick Asylum, Devons  
Road, Bromley-by-Bow, E.
- Laborda, F. E. R., Guy's Hospital.  
Lacey, E. E., I Peterplatz, Vienna.  
Lamb, W. H., M.B., 23, Palace Court, Bayswater Hill, W.  
Lancaster, H. F., M.D., 198, Gloucester Terrace, Hyde Park, W.  
Lancet, The, 423, Strand, W.C.  
Landon, E. E. B., Elderfield Gardens, Kidderminster.  
Lane, W. Arbuthnot, M.S., 21, Cavendish Square, W.  
Langdale, H. M., Ulverston House, Uckfield, Sussex.  
Lansdale, W., M.D., Sunnysdene, Grove Park, Lee, S.E.  
Lansdale, W. M., Guy's Hospital.  
Lansdown, R. G. P., M.D., B.S., 39, Oakfield Road, Clifton,  
Bristol.  
Larkin, F. G., Craven House, Grove Park, Lee, Kent.  
Larking, A. E., M.D., 15, Castle Street, Buckingham.  
Lauder, J. L., Guy's Hospital.  
Laver, C. H., Guy's Hospital.  
Lawrence, F., Guy's Hospital.  
Lawson, F. W., Guy's Hospital.  
Layton, T. B., 48, Queen Anne Street, W.  
Leader, H., M.B., 279, Glossop Road, Sheffield.

- Le Couilliard, F. G., Guy's Hospital.  
 Leigh, H. V., Guy's Hospital.  
 Leigh, W. W., J.P., Treharris, R.S.O., South Wales.  
 Leviser, E. A., Guy's Hospital.  
 Leviser, H. J., Guy's Hospital.  
 Lewis, E. C., Guy's Hospital.  
 Lewis, J. L. D., Guy's Hospital.  
 Lidderdale, F. J., M.D., B.C., 4, London Street, Folkestone.  
 Liebson, A. S., Guy's Hospital.  
 Lindsay, W. J., M.A., M.D., B.C., 84, Herne Hill, S.E.  
 Lipscomb, E. H., M.B., St. Alban's, Herts.  
 Lister, T. D., M.D., B.S., 31, Harley Street, W.  
 Litchfield, E. M., Guy's Hospital.  
 Llarena, E. F., Guy's Hospital.  
 Lloyd, M., M.D., Vale Villa, Llanarthney, R.S.O., Wales.  
 Lloyd, V. E., Guy's Hospital.  
 Lockwood, J. P., Faringdon, Berkshire.  
 Lockyer, G. E., Manor House, Much Wenlock.  
 Long, D. S., B.A., M.D., B.C., 92, Micklegate, York.  
 Long, G. S. B., Guy's Hospital.  
 Loud, F., Albion House, Lewes.  
 Lowe, G. C., 52, Oxley Street, Dockhead, S.E.  
 Lucas, H., Huntingdon.  
 Lucas, R. Clement, B.S., F.R.C.S., 50, Wimpole Street, W.  
 Lucas, R. H., Guy's Hospital.  
 Luce, R. H., B.A., M.B., B.C., 42, Friargate, Derby.  
 Luna, V. A., Pacasmayo, Peru.
- Macalister, G. H. K., B.A., M.D., B.C., Lister Institute, Chelsea  
 Gardens, Chelsea Bridge Road, S.W.  
 MacClymont, C. G., Guy's Hospital.  
 McCarthy, J. McC., M.D., St. George's, Wellington, Salop.  
 McEvedy, P. G., Guy's Hospital.  
 McFarlane, P. S., Guy's Hospital.  
 McGavin, L. H., 32, Weymouth Street, Cavendish Square, W.  
 McKay, W. K., Guy's Hospital.  
 MacLehose, Messrs. James & Sons, 61, St. Vincent Street,  
 Glasgow (2 copies).  
 MacManus, D., Guy's Hospital.  
 Machin, L., Guy's Hospital.  
 Maelzer, N., Guy's Hospital.  
 Maggs, W. A., 14, Upper Wimpole Street, W.  
 Maher, M., Court of Appeal, Cairo, Egypt.  
 Mahor, E. N., Guy's Hospital.  
 Maile, W. C. D., Guy's Hospital.  
 Makepeace, A. J., 22, Warwick Row, Coventry.

- Malcolm, J. D., M.B., C.M., 33, Wimpole Street, W.  
Mann, H. C. C., M.D., B.S., 8, St. Thomas's Street, S.E.  
Manning, T. Davys, M.B., B.S., 2, Gloucester Row, Weymouth.  
Mansell, E. R., 44, Wellington Square, Hastings.  
Marriott, Oswald, M.D., B.S., Hong Kong, China.  
Marshall, R. P., 143, Grange Road, S.E.  
Marshall, W. L. W., Wentworth House, New North Road, Huddersfield.  
Marston, A. D., Guy's Hospital.  
Martin, E. G., Guy's Hospital.  
Martin, J. A., Guy's Hospital.  
Martin, J. N., Guy's Hospital.  
Martin, James P., Slope of the Bank, Box, Wilts.  
Mash, O. N., Guy's Hospital.  
Matcham, Alfred, 116, St. George's Road, Southwark, S.E.  
Mather, H., Guy's Hospital.  
Mathews, H. Dewe, 39, Brook Street, Grosvenor Square, W.  
Matson, R. C., Guy's Hospital.  
May, W. N., M.D., B.S., The White House, Sonning, Berks.  
Mayer, J. W., Guy's Hospital.  
Mayer, R. G., Guy's Hospital.  
Mayston, R. W., M.D., 58, Pier Road, Erith.  
Meachen, G. N., M.D., B.S., 11, Devonshire Street, Portland Place, W.  
Medlock, C. H., Guy's Hospital.  
Meek, J. W., M.D., 329, Norwood Road, Herne Hill.  
Merriman, R. W., M.A., B.Sc., Guy's Hospital.  
Messenger, H. L., Guy's Hospital.  
Metcalfe, G. H., Clare, Suffolk.  
Meyer, Lieut.-Col. C. H. L., M.D., I.M.S., The Ridge, Malabar Hill, Bombay.  
Meyrick-Jones, H. M., M.D., B.S., Overbury, Charlton Kings, Cheltenham.  
Michael, C. E., M.A., M.B., B.C., Trelawne, Crystal Palace Park Road, S.E.  
Millbank-Smith, H. J. M., Worthing Lodge, Worthing.  
Miller, A. A., M.D., B.S., 46, Westminster Palace Gardens, S.W.  
Miller, G. S., Guy's Hospital.  
Millett H., Guy's Hospital.  
Milligan, R. A., J.P., M.D., Ardmae, Northampton.  
Mills, C., P.O. Box 112, Kroonstadt, O.F.S., South Africa.  
Milton, W. T., M.D., M.S., Southernhay, Westmount Road, Eltham, Kent.  
Minett, E. P., M.D., Georgetown, British Guiana.  
Minns, A. N., Guy's Hospital.  
Moberly, A. V., Guy's Hospital.  
Mollison, W. M., M.A., M.C., M.B., Guy's Hospital.

- Montgomery, L. T., Guy's Hospital.  
 Montocchio, J. O. R., Guy's Hospital.  
 Moore, A. G. H., Guy's Hospital.  
 Moore, A. M., 359, High Street, Lewisham, S.E.  
 Moore, G. K., Guy's Hospital.  
 Moore, J. York, Guy's Hospital.  
 Moore, Julius, M.D., Chota Dilkoosha, Enfield.  
 Moore, L. W., Guy's Hospital.  
 Moore, R. A., Guy's Hospital.  
 Moore, W. H., 18, Church Street, Kidderminster.  
 Morgan, E. C. de M., Guy's Hospital.  
 Morgan, J., J.P., Mount Hazel, Pontrhyd-y-Groes, R.S.O.,  
     Aberystwith.  
 Morgan, O. G., Guy's Hospital.  
 Morgan, T., The Colony, Llandovery, Carmarthenshire, S. Wales  
 Morice, C. G. F., M.D., Greymouth, New Zealand.  
 Morrell, F. H., Guy's Hospital.  
 Morris, Arnold, B.A., Clarendon Lodge, Clarendon Avenue,  
     Leamington.  
 Morrish, D. B., Guy's Hospital.  
 Moss, E., M.D., B.S., 6, King Street, Wrexham.  
 Mothersole, R. D., M.D., M.S., 128, St. George's Road, Bolton.  
 Muir-Smith, L., Guy's Hospital.  
 Mullally, G. T., M.B., B.S., Guy's Hospital.  
 Mullins, R. C., M.A., M.B., B.Ch., West Hill, Grahamstown,  
     Cape of Good Hope, South Africa.  
 Munden, C., Ilminster, Somerset.  
 Munro, H. A., M.A., M.B., B.Ch., Lulworth, Rushey Green,  
     Catford.  
 Muriel, G. B., B.A., M.B., B.C., 109, Scotch Street, Whitehaven  
 Murphy, Sir Shirley, 9, Bentinck Terrace, Regent's Park, N.W.  
 Muspratt, C. D., M.D., B.S., Tantallon, Madeira Road, Bourne-  
     mouth.  
 Mutch, N., M.B., B.C., Guy's Hospital.  
 Mutch, R. S., M.D., Hamilton Lodge, Hanger Lane, Ealing, W.
- Naish, G., Beechcroft, Anstey Road, Alton, Hants.  
 Neal, F. J., Guy's Hospital.  
 Neale, B. G., Two Pools, Iron Acton, Gloucestershire  
 Neely, G., Guy's Hospital.  
 Neely, H. B., Guy's Hospital.  
 Nelson, M. K., 12, Belsize Avenue, Hampstead, N.W.  
 Newland-Pedley, F., 22, Willow Road, Hampstead, N.W.  
 Newnham, W. H. C., M.B., 3, Lansdown Place, Victoria Square,  
     Clifton, Bristol.  
 Nicholls, W. H., Guy's Hospital.

Nicholson, J. W., Red Hall, Gainsborough.  
Ninnis, Roger P., Guy's Hospital.  
Noordhoff, P., Librairie Internationale, Groningen, Holland.  
Noott, G., Guy's Hospital.  
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Northampton General Infirmary Library (per the House Surgeon).  
Norton, E. L., The Vicarage, Wingfield, Harleston, Norfolk.

Oates, J. L., Guy's Hospital.  
Odgers, P. N. Blake, B.A., M.B., M.Ch., 16, Castilian Street, Northampton.  
Ogle, C. J., 1, Cavendish Place, W.  
O'Kane, W. J., Guy's Hospital.  
Oliver, C. H., Guy's Hospital.  
Omaha Public Library and Museum, c/o Librarian, Omaha, Nebraska, U.S.A.  
Oram, R. R. W., Cremyll, Bolingbroke Grove, Wandsworth Common, S.W.  
Ordish, F. J., Guys' Hospital.  
Ormond, A. W., 7, Devonshire Place, Upper Wimpole Street, W.  
Osborn, A. G., M.B., Clifton House, Sheet Street, Windsor.  
Ozanne, R. C., Guy's Hospital.

Pakes, A. E. H., B.Sc., 6, Tucker Street, Kimberley, Cape of Good Hope, South Africa.  
Paliologus, A. L., Royal Colonial Institute, Northumberland Avenue, W.C.  
Palmer, A. E., 32, Frederick Street, Loughborough, Leicestershire.  
Palmer, A. S. Morton, M.A., M.D., B.C., 96, Marine Parade, Worthing.  
Palmer, F. W. Morton, M.A., M.D., B.C., 13, Orchard Gardens, Teignmouth, South Devon.  
Palmer, J. Irwin, 47, Queen Anne Street, W.  
Palmer, J. S., Guy's Hospital.  
Pantin, C. S., M.D., B.S., 1, Albert Terrace, Douglas, Isle of Man.  
Parfitt, J. B., Farleigh House, King's Road, Reading.  
Park, W. C. C., 264, Hither Green Lane, S.E.  
Parke, C. J., St. Kilda, Breakspears Road, Brockley, S.E.  
Parker, W. G., M.B., Shirley, Hazeldene Road, Weston-super-Mare.  
Parkes, H. P., Guy's Hospital.  
Parry-Jones, O. G., Guy's Hospital.



- Parry-Price, H., Guy's Hospital.  
Parry, J. H., Guy's Hospital.  
Parry, R., M.B., Ty Newydd, Carnarvon.  
Partridge, A. A. H., M.A., M.D., B.Ch., St. Germans, Grove Road, Sutton, Surrey.  
Partridge, W. L., Guy's Hospital.  
Patey, P. E. H., Guy's Hospital.  
Paul, Frank T., 38, Rodney Street, Liverpool.  
Payne, J. Lewin, 18, Portland Place, W.  
Pearce, D. G., Guy's Hospital.  
Pearce, F. J., 37, Queen Anne Street, W.  
Pearmund, A. S. W., Guy's Hospital.  
Pearson, M., Guy's Hospital.  
Pedley, C. F., Guy's Hospital.  
Pembrey, M. S., M.A., M.D., B.Ch., Guy's Hospital.  
Pendlebury, J. P., Knowles House, Ormskirk.  
Pendred, V., M.D., 2, Lansdown Place, Coventry.  
Penfold, F. W. H., Rainham, Kent.  
Penny, C. H. G., Guy's Hospital.  
Perceval, J. L., Guy's Hospital.  
Percival, G. H., M.B., 66, Abington Street, Northampton.  
Peregrine, H. L., Guy's Hospital.  
Perry, C. E., M.D., 1, Castle Hill Avenue, Folkestone.  
Perry, Sir Cooper, M.A., M.D., Superintendent's House, Guy's Hospital.  
Petley, C. E., Guy's Hospital.  
Phillipps, W. A., M.D., 73, Ashley Gardens, S.W.  
Phillips, C. J., Guy's Hospital.  
Phillips, E. S., Guy's Hospital.  
Phillips, F. B. Willmer, M.A., M.D., B.Sc., 7, Harpur Place, Bedford.  
Pickering, D. V., Guy's Hospital.  
Pickett, L. R., Guy's Hospital.  
Pickett, R. J., Guy's Hospital.  
Pigeon, H. W., M.A., M.C., 13, Albion Street, Hull.  
Piggot, A. P., Kesteven County Asylum, Sleaford, Lincs.  
Pike, Douglas R., B.A., M.B., B.Ch., Stalmine Lodge, Stalmine, near Poulton-le-Fylde, Lancs.  
Pilbeam, L. S., Guy's Hospital.  
Pilkington, F. W., Kencott House, Lechlade, Glos.  
Pillin, H. L., 33, George Street, Hanover Square, W.  
Pinching, Charles J. W., 76, New Road, Gravesend.  
Pitman, K. C., Guy's Hospital.  
Pitt, G. Newton, M.A., M.D., 15, Portland Place, W.  
Plummer, W. E., Wenchow, via Shanghai, China.  
Pochin, C. H. G., Guy's Hospital.  
Pollard, C., M.D., 23, Foregate Street, Worcester.

- Pollard, G. S., Midsomer Norton, Somerset.  
Poole, T. B., M.D., B.S., Laurence House, Valkyrie Road,  
Westcliff-on-Sea.  
Poolman, A. E., B.A., 10, Weymouth Street, Portland Place, W.  
Portway, R. L., Guy's Hospital.  
Poulton, E. P., M.B., B.Ch., M.R.C.P., 21, Belmont Park,  
Blackheath, S.E.  
Prall, S. L., Beechwood, 151, Maida Vale, W.  
Pratt, G. A., Guy's Hospital.  
Pratt, W. H. A., Guy's Hospital.  
Pretorius, W. J., Guy's Hospital.  
Price, John A. P., M.D., 124, Castle Street, Reading.  
Price-Jones, Cecil, M.B., 68, Harley Street, W.  
Pritchard, G. B., Guy's Hospital.  
Pryn, Deputy Surgeon-General W. W., R.N., Royal Naval  
Hospital, Gibraltar.  
Pryn, R. H. C., Guy's Hospital.  
Pryn, W. R., Guy's Hospital.  
Punch, A. L., Guy's Hospital.  
Purdom, H. N., Guy's Hospital.  
Purkis, K. N., Guy's Hospital.  
Puzey, Chauncy, 71, Rodney Street, Liverpool.  
Pye-Smith, C. D., M.B., B.S., Wentworth House, New North  
Road, Huddersfield.  
Pye-Smith, R. J., 450, Glossop Road, Sheffield.
- Quesada, R., Guy's Hospital.
- Rahman, Captain M. A., I.M.S., c/o Messrs. Grindlay, Groom  
& Co., Post Box 93, Bombay.  
Rake, H. V., "St. Ives," Fordingbridge, Salisbury.  
Ramirez, V., Guy's Hospital.  
Ramos, Dr. Alvaro, 76, Rua de Hospicio, Rio de Janeiro, Brazil.  
Ramskill, Josiah, 35, Meadow Lane, Leeds.  
Randall, Philip N., M.B., Tower House, Tweedy Road, Bromley,  
Kent.  
Ransford, L. U., Hamilton Villa, Hamlet Road, Norwood, S.E.  
Ransford, T. D., 6, Queen's Square, Bath.  
Ransford, W. R., c/o Thos. Cook & Son, Ludgate Circus, E.C.  
Ray, Edward Reynolds, 52, Brook Street, W.  
Rayson, H. K., M.D., Knight's Mead, Bayville, Cape of Good  
Hope.  
Reckitt, C. E., Guy's Hospital.  
Reeves, J. K., 66, Upper Tulse Hill, S.W.  
Reid, A., M.B., B.S., Kuala Lumpur, Selangor, Federated  
Malay States.

- Reid, E., 164, St. Helen's Road, Swansea.  
 Reinhold, Captain C. H., I.M.S., c/o Messrs. Grindlay, Groom  
 & Co., 54, Parliament Street, S.W.  
 Reynell, W. R., Guy's Hospital.  
 Reynolds, B. Gore, Oaklands, Montgomery.  
 Reynolds, W. L. E., Guy's Hospital.  
 Richards, D. O., Guy's Hospital.  
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 Richards, L. P., Guy's Hospital.  
 Richardson, J. G. D., 67, Rusthall Avenue, Bedford Park, W.  
 Richardson, W. S., M.D., "Melbury," Christchurch Road,  
 Bournemouth.  
 Ricketts, T. F., M.D., B.Sc., Joyce Green Hospital, Dartford,  
 Kent.  
 Rix, B., 2, Mount Ephraim Road, Tunbridge Wells.  
 Rix, R. H., Guy's Hospital.  
 Roberts, Astley C., Badlesmere, Eastbourne.  
 Roberts, C. S. L., Guy's Hospital.  
 Roberts, H. J., J.P., M.D., Pen-y-groes, North Wales.  
 Roberts, J. Lloyd, B.A., M.D., B.S., B.Sc., 68, Rodney Street,  
 Liverpool.  
 Roberts, R. J., M.A., M.B., B.C., "Upwey," Ventnor, Isle of  
 Wight.  
 Roberts, W. O., 10, Honor Oak Park, S.E.  
 Robertson, C. H., M.B., B.S., Bresna, Alfred Street. Auckland,  
 New Zealand.  
 Robertson, W. I., M.D., St. Annes, Thurlow Park Road, West  
 Dulwich.  
 Robillard, Ete de, Guy's Hospital.  
 Robinson, J. F., 27, Beaumont Street, Oxford.  
 Robinson, W., Guy's Hospital.  
 Robinson, W. E., B.A., M.D., B.Ch., 29, Tedworth Square,  
 Chelsea, S.W.  
 Rodman, G. Hook, M.D., Sheen Gate House, Sheen Lane, East  
 Sheen, S.W.  
 Rogerson, H., M.Sc., Guy's Hospital.  
 Rook, A. F., Guy's Hospital.  
 Rook, H. C., Guy's Hospital.  
 Roots, W. H., Canbury House, Kingston-on-Thames.  
 Roper, A., M.D., Colby, Lewisham Hill, S.E.  
 Routh, C. F., M.D., Holmbush, Grove Road, Southsea.  
 Rouw, R. Wynn, 8, Upper Wimpole Street, W.  
 Rowell, G., 6, Cavendish Place, Cavendish Square, W.  
 Rowland, F. W., M.D., B.S., 3, St. George's Place, Brighton.  
 Rowland, W. J., B.A., M.B., B.S., 5, College Road, Brighton.  
 Rowlands, R. P., M.B., M.S., 12, Queen Anne Street, W.  
 Rowley, A. L., 54, Terrace Road, Aberystwith.

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Russ, S. H., Guy's Hospital.  
Russell, G. H., M.D., Cromwell House, 235, Stockport Road,  
Manchester.  
Russell, J. W., M.A., M.D., 72, Newhall Street, Birmingham.  
Rycroft, A. T., Guy's Hospital.  
Ryffel, J. H., M.A., M.B., B.C., B.Sc., Guy's Hospital.  
Ryle, J. A., Guy's Hospital.  
Ryley, C. M., Guy's Hospital.

Salsbury, A. F., Guy's Hospital.  
Salt, H. O., Guy's Hospital.  
Salter, C. E., M.D., B.S., 34, Prince of Wales Terrace, Scar-  
borough.  
Sampson, B. F., Guy's Hospital.  
Saner, F. D., M.B., B.C., Guy's Hospital.  
Saner, J. G., M.B., B.C., Guy's Hospital.  
Sangster, Charles, 148, Lambeth Road, S.E.  
Saul, E. R., Guy's Hospital.  
Saunders, G. V., Guy's Hospital.  
Saundry, J. Baynard, M.D., 72, Blackheath Hill, S.E.  
Savage, Sir G. H., M.D., 26, Devonshire Place, W.  
Savatard, Louis, 8a, St. John Street, Manchester.  
Saw, N. H. W., Guy's Hospital.  
Scales, J. E., Leigh House, Radstock, Somerset.  
Scott, Arnold, M.D., Bocking, Braintree, Essex.  
Scott, Alfred, 141, Marine Parade, Brighton.  
Scott, B., Stagsden, West Cliff, Bournemouth.  
Scott, D. C., Guy's Hospital.  
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Seabrooke, A. S., Guy's Hospital.  
Selim, A. K., Guy's Hospital.  
Sells, R., Guy's Hospital.  
Sewill, J. Sefton, 9a, Cavendish Square, W.  
Shadwell, St. C. B., M.D., Lynhurst, Orford Road, Walthamstow  
Shaheen, H. I., Shubra Road, Cairo, Egypt.  
Sharpe, H., Guy's Hospital.  
Shattock, C. R., 25, Heathfield Terrace, Chiswick, W.  
Shaw, C. Knox, 19, Bentinck Street, W.  
Shaw, F. H., The Gables, St. Leonards-on-Sea.  
Shaw, Lauriston E., M.D., 64, Harley Street, W.  
Shearwood, A. L., Guy's Hospital.

- Sheen, A. W., M.D., M.S., 2, St. Andrew's Crescent, Cardiff.  
 Sheffield Medico-Chirurgical Society (Dr. R. J. Pye-Smith,  
 F.R.C.S., 450, Glossop Road, Sheffield).  
 Sheldon, T. Steele, M.B., Parkside Asylum, Macclesfield.  
 Sheldon, T. W., Guy's Hospital.  
 Shelswell, O. B., Sibford, Mitcham.  
 Shelton-Jones, E., Pwllheli, North Wales.  
 Sherris, C., Guy's Hospital.  
 Shillitoe, A., B.A., M.B., B.C., 2, Frederick's Place, Old Jewry,  
 E.C.  
 Shipman, George Wm., Grantham, Lincolnshire.  
 Shufflebotham, F., J.P., M.A., M.B., B.C., 21, London Road,  
 Newcastle, Staffordshire.  
 Shute, G. S., M.D., 2, Granby Place, Northfleet, Kent.  
 Sichel, G. T. S., Vine House, Sevenoaks.  
 Siggs, C. G. D., Guy's Hospital.  
 Sigler, Geo. A., M.D., Liberty, Union County, Indiana, United  
 States of America.  
 Simons, G. E. L., Guy's Hospital.  
 Simpson, B. C. W., Guy's Hospital.  
 Simpson, Graham S., Sheffield Royal Hospital, Sheffield.  
 Skelton, W. Bevill, Charges House, Gosport.  
 Small, D. F., Guy's Hospital.  
 Smart, H. D., M.D., B.S., Shelley, near Huddersfield.  
 Smith, A. J. E., Guy's Hospital.  
 Smith, C. R., Guy's Hospital.  
 Smith, G. Bellingham, M.B., B.S., 13, Queen Anne Street, W.  
 Smith, H. L., Guy's Hospital.  
 Smith, S. G., Guy's Hospital.  
 Smith, W. A. Lauder, M.A., M.B., B.C., 8, Chamberlain Street,  
 Wells.  
 Smith, W. H. M., 204, Selhurst Road, South Norwood, S.E.  
 Smyth, W. J. D., Guy's Hospital.  
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 Spon, H. J., 48, Queen Anne Street, W.  
 Spong, R. W., Guy's Hospital.  
 Spurgin, W. H., 8, Graingerville, South Westgate Road, New-  
 castle-on-Tyne.  
 Stainer, C. H., Guy's Hospital.  
 Staley, R. C. W., Guy's Hospital.  
 Stallman, J. F. H., Guy's Hospital.  
 Stamford, R. B., Loughborough, Leicestershire.  
 Stamm, L. E., M.D., B.Sc., 43, High Road, Streatham, S.W.  
 Stanley-Jones, H., M.B., Highlands, Howard Road, Richmond  
 Park, Bournemouth.  
 Starling, E. A., M.B., Chillingworth House, Tunbridge Wells.

- Starling, E. C. W., Guy's Hospital.  
Stebbing, G. F., M.B., B.S., Lambeth Infirmary, Brook Street,  
Kennington, S.E.  
Stebbings, J. M., Guy's Hospital.  
Steinhaeuser, J. R., M.D., B.S., St. Andrew's Place, Lewes.  
Stephens, L. E. W., Emsworth, Hants.  
Stephens, R. F., Methleigh, St. Austell, Cornwall.  
Stevens, J. G., Guy's Hospital.  
Stevens, S., Guy's Hospital.  
Stevenson, J., Guy's Hospital.  
Steward, F. J., M.B., M.S., 98, Portland Place, W.  
Stewart, H. M., M.A., M.D., B.C., Dyffryn, 123, Thurlow Park  
Road, Dulwich, S.E.  
Stewart, R. S., Guy's Hospital.  
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Strover, H. C., Ivel Lodge, Sandy, Beds.  
Sturdy, H. C., M.D., B.S., 81, Bolingbroke Grove, Wandsworth  
Common, S.W.  
Sturges-Jones, W. E., Deloraine, Half-Moon Lane, Herne Hill,  
S.E.  
Sutton, C. R. Arnold, M.A., M.D., B.C., Sidcup, Kent.  
Sutton, J. Spear, Guy's Hospital.  
Sutton, W. H., Guy's Hospital.  
Swan, R. H. J., M.B., M.S., 75, Wimpole Street, W.  
Swayne, F. G., M.A., M.B., B.C., 140, Church Road, Upper  
Norwood, S.E.  
Swayne, W. C., M.D., B.S., Mathon House, 56, St. Paul's Road,  
Clifton, Bristol.  
Symonds, Charters J., M.D., M.S., 58, Portland Place, W.
- Ta'Bois, F. W., 2, Mornington Villas, Woodford Green, Essex.  
Tanner, John, M.D., 8, Glenmore Road, Belsize Park, Hamp-  
stead, N.W.  
Tanner, W. E., Guy's Hospital.  
Targett, J. H., M.S., 19, Upper Wimpole Street, W.  
Taylor, Arthur S., M.D., Lovelace Lodge, Lovelace Road,  
Surbiton.  
Taylor, E. S., Guy's Hospital.  
Taylor, Frederick, M.D., 20, Wimpole Street, W.  
Taylor, H. Owen, M.D., Oxford Street, Nottingham.

Taylor, J. G., M.A., M.D., B.C., Grey Friars House, Chester.  
 Taymour, A. H., Guy's Hospital.  
 Tehbitt, E. R., 4, Frant Road, Tunbridge Wells.  
 Telling, W. H. Maxwell, M.D., B.S., 29, Park Square, Leeds.  
 Thavara, M. C., Guy's Hospital.  
 Thin, Ba-, Guy's Hospital.  
 Thom, M. C. V., Guy's Hospital.  
 Thomas, A., M.B., B.S., North Parade, Aberystwith.  
 Thomas, C. E., Guy's Hospital.  
 Thomas, F. L., M.B., B.S., The Square, Barnstaple, N. Devon.  
 Thomas, T. P., B.A., Bank House, Brecon.  
 Thompson, A. R., M.B., Ch.M., 49, Wimpole Street, W.  
 Thompson, H. Q. F., Guy's Hospital.  
 Thompson, W. A., Guy's Hospital.  
 Thomson, D., Ulverstone, Devon, Tasmania.  
 Thorpe, W. G., M.D., 270, Balham High Road, S.W.  
 Ticehurst, N. F., B.A., M.B., B.C., 35, Pevensey Road, St.  
     Leonards-on-Sea.  
 Tilbury, A., Guy's Hospital.  
 Tilbury, R., 235, Queen's Road, Peckham, S.E.  
 Todd, G., Sydenham, Torquay.  
 Tongue, E. J., Winterton, Doncaster.  
 Tooth, F., Guy's Hospital.  
 Totton, J., Guy's Hospital.  
 Townsend, T. A., Guy's Hospital.  
 Trail, D. H., M.B., Darjeeling, Woodlane, Falmouth.  
 Traill, K. B., Guy's Hospital.  
 Trethowan, W. H., M.B., B.S., Guy's Hospital.  
 Trounce, T. R., Guy's Hospital.  
 Turner, F. Douglas, M.B., Eastern Counties Asylum, Colchester.  
 Turner, H. A., Ely Lodge, Lismore Road, Eastbourne.  
 Turner, H. Gunton, Holmwood, Bournemouth.  
 Turner, H. S., Fox Bay, Falkland Islands, S. America.  
 Turner, P., M.B., M.S., B.Sc., Guy's Hospital.  
 Turner, T., M.B., B.S., 1, Hardwicke Road, Eastbourne.  
 Tweed, M. B. W., Guy's Hospital.  
 Tyson, W., M.A., M.D., B.C., The Beeches, Lowestoft.

Uththoff, John C., M.D., Wavertree House, Furze Hill, Hove,  
Sussex.

University College Library, London, Gower Street, W.C.

University College Library, Dundee.

Valerio, I., San José, Costa Rica, Central America.

Vance, W. J., Guy's Hospital.

- Veale, R. M., Guy's Hospital.  
Venables, J. F., Guy's Hospital.  
Victory, J., Guy's Hospital.  
Vidot, S., Guy's Hospital.  
Viljoen, W. J. R., Guy's Hospital.  
Viney, J. E., M.A., M.D., Harcourts, Chertsey.
- Wacher, F., Castleden Lodge, Canterbury.  
Wacher, H., B.A., M.B., B.C., 72, St. Dunstan's Street, Canterbury.  
Wacher, H. S., Guy's Hospital.  
Wacher, S., St. George's Place, Canterbury.  
Waddy, H. E., Rhossili, Brunswick Road, Gloucester.  
Waghorn, L. P., Guy's Hospital.  
Wainewright, R. S., M.D., 7, Grand Avenue, Hove, Sussex.  
Waite, D. A., M.A., Bel Air, Woodstock Avenue, Golder's Green, N.W.  
Wakefield, C. F., Waywell, Norwood Hill, Charlwood, Surrey.  
Wales, T. Garneys, Downham Market, Norfolk.  
Walker, T. M., Roseneath, Hailsham, Sussex.  
Waller, W. A. E., Colonsay, Rugby.  
Wallis, M. J. T., Guy's Hospital.  
Ward, E. L., 32, Wellington Street, Merthyr Tydvil.  
Warner, H. P., M.B., B.S., Royal Hants County Hospital, Winchester.  
Watkin, P. J., Guy's Hospital.  
Webb, W. L., Guy's Hospital.  
Webster, E. M., Guy's Hospital.  
Welchman, F. E., 16, Carlton Road, Putney, S.W.  
Welton, F. E., Guy's Hospital.  
Westman, K., Guy's Hospital.  
Wheeler, F. J., 95, Cambridge Road, Southport, Lanes.  
Wheldon, G. W., Guy's Hospital.  
White, W. Hale, M.D., 38, Wimpole Street, W.  
Whitten, M. G., Guy's Hospital.  
Whitworth, H. P., Guy's Hospital.  
Whitworth, W. C., Guy's Hospital.  
Wickenden, S., Guy's Hospital.  
Wilkins, J. C. V., Locksley, Sarisbury, near Southampton.  
Wilkinson, H. B., Plymouth Borough Asylum, Blackadon, Ivy-bridge, Devon.  
Willan, G. T., junr., 84, Crown Road, Milton, Sittingbourne.  
Williams, Fleet-Surgeon E. H., c/o Dr. MacDougal, 9, East Park Terrace, Southampton.  
Williams, Edgar R., Guy's Hospital.  
Williams, W. C. H., Guy's Hospital.



- Williams, W. E., Guy's Hospital.  
 Wills, A., Guy's Hospital.  
 Wilson, A. R., B.A., M.D., B.Ch., "Entfield," Old Town, Bexhill-on-Sea.  
 Wilson, S., Guy's Hospital.  
 Wilson, W., M.B., C.M., 184, Goldhawk Road, Shepherd's Bush, W.  
 Wilson-Smith, T., M.D., 26, The Circus, Bath.  
 Windermere, L. D., Guy's Hospital.  
 Wohlmann, A. Stanley, M.D., B.S., Rotorua, New Zealand.  
 Wood, P. M., Redcliffe, Liverpool Road, Ashfield, Sydney, N.S.W.  
 Wood, T. A. V., Guy's Hospital.  
 Wood, T. N., Woodlands, Gravesend.  
 Woodroffe, B. C., Guy's Hospital.  
 Woodruff, K. M., Guy's Hospital.  
 Woodward, H. M. M., St. John's Hospital, Lewisham, S.E.  
 Woolf, S. A., Guy's Hospital.  
 Wornum, G. Porter, 58, Belsize Park, Hampstead, N.W.  
 Worthington, H. E., The Sycamores, Birchington-on-Sea.  
 Wright, H. H., Ospringe Road, St. John's College Park, N.W.  
 Wright, J. E., Guy's Hospital.  
 Wright, L. D., Guy's Hospital.  
 Wylie, Angus, M.A., M.B., B.C., 3, Regent's Park Road, N.W.
- York Medical Society (care of Dr. Northcote, Blenheim House, Monkgate, York).  
 Young, F., Guy's Hospital.  
 Young, F. C., B.A., M.B., B.C., Meadowside, Twyford, Berks.  
 Young, John, M.D., 94, Stamford Hill, N.  
 Young, W. A., Guy's Hospital.

Zamora, A. M., Guy's Hospital.

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Revue de Médecine (Monsieur le Docteur Lepine, 30, Place Bellecour, Lyons)

Royal London Ophthalmic Hospital Reports, City Road, E.C.

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**Westminster Hospital Reports**



Univ. of  
California

TO VIRU  
AMPHOTIAO



*F. W. Pavy*

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*Born 1829*

*Died 1911.*

## Memorandum

ADAM WILLIAM FAVY.

**PASS BY REFERENCE**

Journal of Management Education 29(1): 1-12

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*F. W. Pary*

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2nd 1900

## In Memoriam

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### FREDERICK WILLIAM PAVY,

M.D., F.R.S., F.R.C.P.

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FREDERICK WILLIAM PAVY was born at Wroughton, near Swindon, in Wiltshire, on May 29th, 1829.

His father, William Pavy, was of French extraction, and came to England in the early part of the nineteenth century. A near relative of William Pavy became Archbishop of Algiers. In a biographical memoir of this distinguished prelate it is stated that the Pavy family originated in Savoy, and was well represented in the neighbourhood of Chambéry; but the branch from which the Archbishop was descended had been for many years settled in the town of Roanne, in the department of the Loire; and a great uncle of his, François Pavy, was haled before the Revolutionary Tribunal at Lyons in 1793, being vicar in that town at the time. Louis Antoine Augustin was born in 1805, and was made Bishop of Algiers in 1846. He laid the foundation stone of Nôtre Dame de l'Afrique in Algiers in 1855, and saw the completion of the cathedral. He was created Archbishop in 1865, but died the following year. It is interesting that General Pelissier, who commanded the French troops in the Crimea after the resignation of General Canrobert, was intimate with Monseigneur Pavy, and presented to Nôtre Dame de l'Afrique the sword which he had carried at the successful attack on the Malakoff.

Dr. Pavy's father, William Pavy, appears to have been born in 1797. He, also, was destined for the church, but an open-air life appealed to him; he migrated to England as a comparatively young man, took up the business of a maltster, and bought an estate of about 200 acres at Wroughton. Here he built Woodham House, in which Dr. Pavy was born.

William Pavy married Miss Pickett, the youngest daughter of a landholder in the neighbourhood, and had nine children—four sons and five daughters. Within a few months of the birth of the youngest son, the father died at the age of 39.

This youngest son, Francis, had a successful career. He desired a military life, and at the age of eighteen volunteered for service in the Crimea, and arrived at Scutari just after the battle of Inkerman, but he caught fever, and was compelled to return home. Two years later he was gazetted to the 74th Highlanders, and served with distinction in the Indian Mutiny. He became a captain and retired on half-pay. He then became an accountant, and soon took a leading position, becoming director and chairman of several important companies. Captain Pavy was also a member of the Honourable Corps of Gentlemen-at-Arms. He lived at Foley House, Portland Place, and died in 1902.

One member of this family, Dr. Pavy's last surviving sister, Mrs. Fisher, still resides at Elcombe Hall, a property near Wroughton, which was also in the possession of her father, William Pavy. The writer is indebted to her for much kindness and help in regard to this memoir.

Frederick William was the second son. Independence of character in early life is always an interesting feature in those who are afterwards distinguished, and this independence was shown by young Pavy; for, being sent to a preparatory school at Swindon, he was so displeased or unhappy, that he ran away home, and, being taken back, again effected his escape.

After this he was sent to Merchant Taylors' School in London in the month of March, 1840. He began in the petty form, was moved into the first form six months later, into the

second form in March, 1841, into the third form, March, 1842, and was in the division form from October, 1842, to March, 1843, when he seems to have left.

Exactly what he did in the next few years is not quite clear; but the most interesting fact is that though he seems to have desired to become a doctor, he was not at first put in the way of studying for a medical career. When the writer of this article was a student, an occasional visitor at his father's house was one of the old school of business men in the city, a ship-broker, Mr. Woollett. He was intimate with Mrs. Pavy, and he and his wife paid her a visit at the house near Swindon every year. When young Pavy left school Mr. Woollett was asked to take him into his office in Lime Street Square in order to see how he would like a commercial life. How long he was there it has not been possible to ascertain, but, at any rate, the life was not congenial to him, and he gave it up. Some part of the time between school and hospital he seems to have spent on his father's estate, and he actually contemplated farming as an occupation; probably this was after his City experience, but, in any case, it was also abandoned.

The next event in his history of which one can be sure is his entry at Guy's Hospital, which occurred in May, 1847, four years after his leaving school. He then, probably with a view to taking the London University degree, and perhaps because he may have been reading for the Matriculation examination, took out the course of botany alone, and in October of the same year paid further fees, but was not really entered as a first year's student until 1848. It was also not until this year that he passed the Matriculation of the London University, being entered in the register as from Merchant Taylors' School. He took honours in Chemistry.

It may be interesting to old Guy's men to note the names of some of those who were his contemporaries at the date of his complete entry in 1848. Amongst third year's men we see the names of W. Odling, Thomas Bryant, C. D. Kingsford, Sherwood Stocker, R. Growse, and T. A. Waterworth. Amongst second

year's men: A. T. Brett, S. Knaggs, Morley Rooke, Buchanan Washbourn, and Jas. Bisshopp. Amongst first year's men: John Wilton, Constantine Holman, and J. Ramskill.

Pavy had a successful career as a student. It must be remembered that sixty years ago neither the examinations nor the hospital appointments were quite the same as they are now. The preliminary scientific examination of the University of London, now known as the first examination for medical degrees, was not instituted until 1861. The first examination in the medical curriculum was the First M.B., and this Pavy passed in 1850, being placed in honours, third in Anatomy and Physiology, third in Chemistry, fourth in Botany, and obtaining the Exhibition and Gold Medal in *Materia Medica*.

At Guy's Hospital he was full dresser to Mr. Hilton in April, 1851, and resident obstetric clerk in June of the same year.\* In 1852 he obtained the degree of Bachelor of Medicine, being placed in honours, third in Physiology, third in Surgery, second with the Gold Medal in Medicine, and alone in Midwifery. In these honours examinations Pavy was run close by another Guy's man, Morley Rooke, afterwards in practice at Cheltenham; Rooke indeed took the Scholarship and Gold Medal in Medicine, was bracketed with Pavy in Physiology, but was below him in Surgery. On this occasion the Scholarship and Gold Medal in Surgery were taken by Joseph, afterwards Lord, Lister.

It only needs a glance over the honours lists of those days to refute the belief, entertained so conveniently by some, that men who distinguish themselves at their degree examinations are bound to be failures in after-life.

Pavy obtained the degree of Doctor of Medicine at the University of London in the succeeding year, 1853; but in the interval between his M.B. and M.D. degrees he had already shown his interest in the relations of sugar to the human economy, had come into contact with the distinguished French

\* It has been stated that he was both House Surgeon and House Physician. This is an error. The former appointment was only instituted in 1856, and the latter in 1868.

physiologist, Claude Bernard, and had commenced those researches and that devoted study of this difficult subject which, we might say, he never abandoned for one moment of the remaining fifty-eight years of the life he was destined to enjoy.

His first contribution to the Guy's Hospital Reports was in 1853.\* It is entitled "Saccharine Matter: its physiological relations in the economy;" by Frederick William Pavy, M.B. London. In a footnote he writes: "In making these succeeding remarks I feel bound to state that my information has been principally derived from the opportunity I have had, during three months of the past summer, of assisting M. Bernard in his experimental investigations at the laboratory of the Collège de France." And then he acknowledges his indebtedness to the professor for his courtesy and ready help. This, then, must have been the summer of 1853.

Pavy's distinguished career as a student could not fail to give him an opportunity of joining the teaching staff, and that his bent was towards physiology is shown by the fact of his work in Claude Bernard's laboratory. For a time he was Demonstrator of Anatomy, but we find him in the session of 1856-57 lecturing on Physiology in the winter and on Comparative Anatomy in the summer. His predecessor in Physiology had been Dr. Gull. He held this double lectureship until 1865, when he resigned the summer lectures to Dr. Pye-Smith; but he continued to lecture on Physiology until 1877. For the greater part of these 22 years he gave the full winter course of six months' lectures on this subject; but in the last five years Dr. Pye-Smith was associated with him, taking one-half the winter lectures.

On leaving the chair of Physiology, he joined Dr. Wilks in the course of lectures on Medicine; but these he delivered only for six years, resigning the systematic lectures on Medicine five or six years before his clinical work as a physician was brought to a close.

\* Guy's Hospital Reports, Second Series, vol. viii., 1853.

It was not until November, 1858, when Pavy was in his thirtieth year, and had already been teaching for four or five years in the Medical School, that the chance of an appointment on the active staff of the hospital was offered to him. This occurred through the death of Dr. Hughes.

His seniors on the Physicians' Staff were then Drs. Addison, G. Hilario Barlow, Owen Rees, and Gull, as full physicians, and Drs. Habershon and Wilks as assistant physicians. In the spring of the year 1860 Dr. Addison retired from the hospital, and died a few months later; but no fresh appointment was made, and the medical staff consisted of three physicians having wards and three assistant physicians seeing out-patients. In 1871 the northern half of the present medical buildings, called Hunt's House, was completed, and this added 120 beds in three wards, which were named after Astley Cooper, Bright, and Addison.\* This large increase in medical beds necessitated the appointment of another full physician, and Dr. Pavy was promoted from the rank of assistant physician, having for his seniors Drs. Owen Rees, Habershon, and Wilks.

The law of superannuation, applied to the medical and surgical staff, which should have operated in Dr. Pavy's case in 1889, was most inopportune. Death had played havoc in the ranks of the medical staff in recent years. Hilton Fagge had died six years previously, Mahomed a year after Fagge, an interval of only two years and Moxon ended his brilliant career, to be followed in less than twelve months by Carrington. After this last sad event in the opening of 1887, the assistant physicians,

\* These wards on the first, second, and third floors of the northern division of Hunt's House have had different names from time to time in accordance with varying conditions of the hospital finances, which required at one time the dismantling of two or three wards, and with the need of expansion on the surgical side, met by the conversion of one ward into small operating theatres. They are now named respectively Mary, Esther, and Queen Victoria wards, and the old names have been transferred as follows: Astley Cooper, to a surgical ward (formerly Accident); Bright, to the private ward for paying patients on the third floor of the southern division of Hunt's House (originally Mary ward); and Addison, to the male ward on the first floor of the southern division (originally Philip).

who had been working at half strength for some time, were brought up to their full number by the appointment of three more. But Fate had not yet done with the Guy's Medical Staff, for within a week of the normal period for the retirement of Dr. Pavy from the Staff, that able physiologist, Leonard Wooldridge, whose career had begun with so much promise, and who was one of those appointed assistant physician just two years previously, was struck down by fatal syncope. The Governors, upon hearing this, while nominating Dr. Lauriston Shaw to fill Dr. Wooldridge's place, asked the Treasurer to invite Dr. Pavy to retain his appointment for another twelve months from that date.

Thus, it was only in 1890 that Dr. Pavy joined the consulting staff, and so far as teaching, clinical or otherwise, was concerned his work within the walls of Guy's Hospital was finished.

As a lecturer Pavy was clear and precise, and in simple language put the facts lucidly before his audience. No doubt he was at his best in the subject of Physiology in which he was an original worker and experimenter; and as his lectures on this subject were almost entirely before the passing of the Vivisection Act of 1876, he was not hampered by legal restrictions if he wished at any time to give his students that best of all opportunities of learning the facts of nature, an ocular demonstration. He was a very clever operator, and the neatness with which he used the scalpel in exposing the anatomy of the animal under experiment gave everyone who saw him the impression that he would have made an excellent surgeon. One lecture especially lives in the memory of the writer. It was delivered in the session 1864-65, and was intended for publication, with the object of refuting the statements of Claude Bernard with regard to the amount of sugar to be found in the blood of the hepatic vein. Pavy complained that his results had not been accepted by other observers, but triumphantly pointed out that Dr. McDonnell had obtained the same results in experiments which, for better confirmation, were made upon cats, and not upon dogs. "Now," said Pavy, with much glee, alluding to the defensive



mechanisms enjoyed by this "harmless, necessary" quadruped, "the cat, gentlemen, is not an animal that my experience has induced me to show a partiality towards for physiological experiment."

As a lecturer, in his early days at least, when addressing the students of the first and second years, he had a somewhat nervous manner, which he was apparently trying to overcome with some effort; and this was often the occasion for an exaggerated applause on the part of his audience, both on his entry to the theatre and at other times. And the more cheery spirits amongst the students not infrequently took the opportunity of making disturbances which were, on a few occasions, with difficulty controlled. Still, his lectures were well attended, and highly appreciated by the students; and there is no doubt that his fame as a physiologist was alone sufficient to attract some students to the otherwise rather unfashionable district of "the Borough" for their medical education.

In his teaching in the out-patients and in the wards Pavy did not aim at elaborate disquisitions on the case in all its possible bearings or spend much time over pathological theories, or on disputed points of pathogeny, or treatment. He dealt consistently with the case before him, with its diagnosis and its treatment. He had also this merit, which everyone of his colleagues could not claim, that he was always punctual and spent a proper and reasonable time over his cases without being hustled away for important consultations elsewhere.

On his retirement from the active work of the hospital, Dr. Pavy was presented by the students of the hospital with a handsome silver plate; the presentation took place in the Anatomical Theatre. A fortnight later he was entertained at a Complimentary Dinner at the "Ship," Greenwich, with Mr. Arthur Durham in the chair.

Dr. Pavy took an active part in the working of the Royal College of Physicians. In 1856 he became a Licentiate of the College under the arrangements in force prior to the Medical Act of 1858, when there were no Members; but he was made

one of the Members of the College on the creation of that order in 1859. He was elected a Fellow in 1861, and delivered the Goulstonian Lectures on "Assimilation and the influence of its defects on the urine" in the following year. And in due course he was examiner in physiology, examiner in medicine, a member of the Council, censor and senior censor, the last in the session 1891-92.

He was a frequent lecturer at the College. In addition to the Goulstonian Lectures, he delivered in 1878 the Croonian Lectures "On certain points connected with diabetes." In 1886 he was Harveian Orator, and directed his remarks towards illustrating the value of experimental research by a reference to recent work in the then comparatively new sphere of bacteriology; and in 1891 he was a second time Croonian Lecturer, his subject being "A new departure in connection with diabetes."

Six years later he delivered a supplementary Croonian Lecture on "Points connected with the pathology and treatment of diabetes"; and finally, in 1907, at the age of 78 he gave three lectures on "The pathology and treatment of diabetes viewed by the light of present-day knowledge."

In 1901 the College of Physicians awarded him the Baly Gold Medal for Distinction in the Science of Physiology.

He was an active member of the various London Medical Societies, and was at different times President of the Pathological Society and of the Royal Medical and Chirurgical Society. Before the Medical Society he delivered the Lettsomian Lectures on "Certain points connected with diabetes" in 1860.

It is now time to say something of Pavy's life's work, that is, his researches and writings upon the behaviour of sugar and other carbohydrates in the animal economy, and especially the morbid appearance of sugar in the urine usually known as diabetes. Pavy's work is, perhaps, the most remarkable instance of steady persistence in one relatively limited line of inquiry, and illustrates not only the extraordinary tenacity of purpose of the inquirer, but also, and in no less degree, the evasiveness and the almost complete insolubility of some problems of life and disease.

Pavy devoted little short of 60 years of his life to this particular problem, for he was working with Bernard in the summer of 1853, and he was still continuing his laboratory experiments up to the time of the autumn holiday just preceding his death in 1911.

There was remarkable sagacity, not devoid of satire, in Gull's comment upon Pavy. What sin, said he, has Pavy committed, or his fathers before him, that he should be condemned to spend his whole life seeking for the cure of an incurable disease? And Pavy, still researching, survived Gull by some one and twenty years.

It is not desirable to attempt anything like a review of the subject of diabetes during the last 50 years, even as it presented itself to Dr. Pavy, but a brief account of his work in this direction may be allowed. His first printed article upon the subject appears to be that already mentioned as being published in the eighth volume of the Second Series of the Guy's Hospital Reports entitled "*Saccharine Matter, its physiological relations in the animal economy.*" He had worked in Claude Bernard's laboratory and he had heard his lectures at the *École Pratique*. It appears also that he had himself performed experiments and made observations on the relation of sugar to the liver within the walls of Guy's Hospital, even before going to Paris. At any rate, this article, published in 1853, refers to numerous experiments conducted by himself in confirmation and extension of Bernard's work.

In the same year he must have made a communication to the Royal Society, for there is an interesting note in the first volume of the Third Series of the Guy's Hospital Reports, 1855, which is to the following effect:—"Among the distributions of the Scientific Grant made to the Royal Society for the year 1854 occurs the following: 'No. 12. To Dr. Pavy, for continuing experimental researches on the physiology of the blood, of which a part has been recently communicated to the Royal Society, £50.' The researches are in progress."

He published also in Paris in 1854 a pamphlet of some sixteen pages entitled "*Recherches sur la destruction du sucre normal dans l'économie animale.*" The paper had been read before the Société de Biologie on August 26th, 1854, and it referred to the conditions under which sugar appeared to be destroyed by oxidation in the blood.

During the next few years, that is, up to and including 1861, the Proceedings of the Royal Society contain six papers by him on the changes of sugar in the system, the alleged sugar formation of the liver, the lesions of the nervous system producing diabetes, and the influence of acids and of alkalies upon the saccharine state. This work is represented in the Philosophical Transactions only by one paper, which was read in June, 1860, entitled "*Researches on sugar formation in the liver,*" an abridgment of a paper presented in 1858 with some additional matter; but his work over these ten years gained for him admission as a Fellow of the Society in 1863.

Perhaps the most interesting of these communications is that in which he broke away from Claude Bernard, and asserted that the blood of the right heart did not, as Bernard supposed, and Pavy himself till that date, 1858, had believed, contain sugar in any material quantity during life, but that the quantity usually found in it was due to a rapid formation of this substance after death.

These observations were not readily accepted, and we find him in 1865 re-asserting his position in the lecture already referred to,\* again, in 1875, in Communications to the Royal Society, and even again so recently as 1908 in the special course of lectures which he delivered before the Royal College of Physicians. In another of these articles he recorded his experiments confirmatory of Claude Bernard's puncture-diabetes, with his own original observation, that glycosuria was also produced by removal of the superior cervical ganglion, or by division of the sympathetic cord in the neck.

\* *Medical Times and Gazette*, 1865, vol. i.

In 1862 appeared his book entitled "Researches on the nature and treatment of Diabetes," a volume of 168 pages, excluding an appendix of cases; and in 1869 a second edition appeared, of which the subject matter is more than half as much again as in the first.

In the ten years from 1875 to 1884 he was again an active contributor to the Proceedings of the Royal Society, writing on the production of glycosuria by the action of oxygenated blood upon the liver, on the physiology of sugar in relation to the blood, on the physiology of the carbohydrates in general, and on a useful modification of the quantitative test for sugar. Throughout he was consistently and persistently fighting the glycogenic theory of Bernard, not, that is, that he doubted the existence of the substance called glycogen, nor the fact that glycogen is formed in the liver, but he denied the statements that the liver habitually produces large quantities of sugar by a continuous transformation of its previously formed glycogen (sugar producer), that is, he denied the sugar-forming function of the liver, and all that pertained to such a belief. Thus, he showed that during life no change of glycogen into sugar took place in the liver; that oxygen does not destroy the sugar in the blood; that glycogen exists itself in the blood.

With that conviction he regarded the word glycogen as a misnomer, and proposed more than one substitute which, he thought, should be rather a ticket than a name connoting any natural relation. Thus, *amyloid substance* was one term used by him, *hepatine* was another, and in 1881 he proposed to call the substance after its own discoverer, *Bernardine*. But circumstances have been too strong for him, and the name glycogen still remains.

The fact that glycosuria might persist in severe cases, even when no carbohydrate were ingested, led to the recognition of a disorder of metabolism much more serious than the mere incapacity to assimilate superfluous starch or saccharine matters. And Pavy used the terms *alimentary* and *composite*, the former for the simpler and more tractable form, the latter for the variety in which the metabolic disturbance was more complex.

In connection with the practical methods for the diagnosis of diabetes and glycosuria, Pavy's name will be long remembered. At Guy's Hospital *Pavy's blue fluid* was the test commonly used as the equivalent of Fehling's solution. It differs from Fehling's in containing a larger proportion of alkali (17 : 10), which Pavy preferred, because it prevents precipitation by the small quantity of reducing substance present in normal urine. It is interesting to note that the test solution used in Bernard's laboratory, and by Dr. Pavy in his early work, was known as *Barreswil's solution*. This is a solution very similar to Fehling's, containing almost the same amount of cupric sulphate, much less caustic potash, and somewhat more of a mixture of potassium bitartrate or sodic carbonate.

Pavy added to the accuracy of the cupric quantitative test by the use of an ammoniacal solution, whereby the cuprous oxide was held dissolved and the recognition of colour change was greatly facilitated.

A third useful idea was represented by *Pavy's pellets*, by which in the very earliest of days of the history of tabloids, the two elements of Fehling's solution were separately produced in a solid compressed form, requiring solution only at the moment of applying the test.

Pavy's later researches on the physiology of the carbohydrates and their relation to diabetes and glycosuria were directed to ascertaining how the ingested carbohydrates are disposed of and assimilated, if they are not, as Bernard supposed, simply stored up in the liver, to be paid out as sugar and chemically destroyed.

In 1893, in his paper on "The Glucoside constituent of proteid matter" he produced the key to the problem. He showed that a substance nearly identical with sugar could be obtained by the prolonged action of potash upon proteid, and this gave the clue to the occurrence of those cases or stages of diabetes in which glycosuria continued to occur, although no carbohydrates were being ingested. But the important outcome of this was the converse position, viz., his demonstration that carbohydrates in the alimentary canal could, on the one hand, be combined,

probably by the agency of the cells of the villi, with nitrogenous material (peptones) to form proteid matter, and, on the other, could be by the same agency transformed into fat. Consequently, not only was the liver an organ which in its production and retention of glycogen prevented sugar from reaching the general circulation, but the intestinal villi and their related cells formed an even earlier line of defence by their prompt utilisation of the carbohydrates for the formation of those essential substances, proteid and fat. Failing these barriers to the passage of soluble carbohydrates, glycosuria must result. To the development of this theory Pavy devoted the remaining years of his research life.

Pavy's labours were not, of course, confined to diabetes. He did good work in connection with albuminuria, with the physiology of digestion, and with the study of food and dietetics. Moxon had described a remittent and intermittent albuminuria; Pavy described in 1885 a cyclical albuminuria, now usually known, in part at least, as postural albuminuria. He had great confidence in the ferrocyanide test for albumen, and advocated for practical convenience in its application the use of pellets of potassium ferrocyanide and of the necessary citric acid.

In connection with the physiology of digestion his valuable paper on the "Immunity enjoyed by the Stomach from being digested by its own secretion during life" gained admission to the Philosophical Transactions in 1863, and in 1867 he published a "Treatise on the Function of Digestion, its disorders and their treatment."

In 1874 appeared a "Treatise on Food and Dietetics, physiologically and therapeutically considered," and a second edition of this was issued in the following year.

In 1876 Dr. Pavy engaged in some researches upon food metabolism, and urinary excretion as affected by exercise. An American, E. F. Weston, was performing unusual feats of pedestrianism, in covering 500 miles or 1000 miles in as many consecutive hours; and careful comparisons of the food taken and the amount of the urinary constituents before, during, and after

the period of exertion was made by Dr. Pavy, and published in the *British Medical Journal*.

Pavy was soon recognised by the profession and the public as an authority on the above different subjects, but especially upon diabetes; and he had an extensive practice, which continued to the end, for even a few days before his death he was seeing patients at his house. For the first few years of his professional life Dr. Pavy was resident in Finsbury Square, that old home of the City doctor, now passing rapidly into the hands of the City merchant. In 1858, however, he had moved to 33, Bedford Place, Russell Square, and he was there for about seven years, when he went to 35, Grosvenor Street, the house in which he remained till the end.

It is remarkable how he found time for the extensive researches which he conducted. His consulting room in Grosvenor Street was adorned with a beautiful fernery occupying the whole of the south side of the room, but the initiated knew that behind this was a laboratory in which an assistant was testing the urine while the patient was describing his woes to the physician. Pavy, however, always had the use of a more complete installation. As long as he was lecturing on Physiology he had the laboratory in connection with the theatre. When he retired from that post he was accommodated by the Governors with a small room just to the south of the grand entrance of the medical building. This he had to resign when he retired from the active staff, and the room was used as a clinical laboratory by the Registrars and Medical Ward Clerks.

Pavy then took refuge in the laboratories of the Royal Colleges of Physicians and of Surgeons in their building on the Victoria Embankment, near Waterloo Bridge.\* These laboratories were opportunely opened in 1890, and Dr. Pavy was one of the first to receive permission to make use of them for his ex-

\* In April of the present year, 1912, this building was handed over entirely to the Institute of Electrical Engineers, and the Royal Colleges transferred the examinations to their new building in Queen Square, Bloomsbury.



periments. Here almost any afternoon about four or half-past four o'clock might be seen, waiting for Dr. Pavy, his well-known carriage of snuff-brown, picked out with red, and his fine pair of black horses which, he told the writer, he always bought himself at Tattersall's. Pavy's last physiological resting-place was not to be on the Embankment, for, in 1903, the laboratories were closed to private members of the profession and devoted to more public uses, such as the preparation of diphtheria antitoxin for the Metropolitan Asylums Board, the researches carried out under the Cancer Research Fund, and the pathological work of the Royal Army Medical College.

Happily, just before these changes on the Embankment the University of London established a centre for physiological research in its new home in the eastern wing of the Imperial Institute. In January, 1902, the senate adopted a scheme for the institution of University Lectures on Physiology in association with a lecture room and a research laboratory. The equipment for the laboratory was provided by a donation of £2,000 from Sir Walter Palmer, and by contributions from others, among whom was Dr. Pavy himself. The laboratory was opened in November, 1902, by the Chancellor, Lord Rosebery, and Dr. A. D. Waller was appointed Director. Here for the last nine years of his life Dr. Pavy conducted his experiments, making daily visits as he had always regularly done. He was one of the first panel of lecturers appointed in connection with this scheme, and he delivered in 1905 the lectures on "Carbohydrate Metabolism." In 1909 and 1910 he was working at intracellular enzyme action, and in the following year up to the end of the summer session he was continuing his researches into carbohydrate metabolism. Many of the results of the work done in this laboratory were published in the *Journal of Physiology*.

As medicine is, after all, the art of healing, it should be of interest to us to know how Dr. Pavy dealt with the treatment of diabetes. The writer was, for a few years, Medical Registrar, thus assisting in the reporting of the medical cases, and for several years he was Assistant Physician to Dr. Pavy. Though he

could not escape the impression that Dr. Pavy's cases of diabetes in the hospital were not more amenable to treatment than those of other physicians, it must, of course, be remembered that the cases coming into a general hospital are nearly always, pathologically, the worst type of case, and that individually patients of the hospital class are most unwilling to submit to the discipline of dietetic treatment, which they will evade by the most deceitful methods. Moreover, there was very little variety in the methods of treatment at that time; dietetic restrictions were generally employed, and the efficacy of drugs was not much more trusted than it is now. Still, there can be no doubt that Dr. Pavy must have been successful in dealing with that numerous class of cases which he called alimentary diabetes, in which among middle-aged patients with a small amount of glycosuria, are kept in good health by an adequate restriction of their carbohydrate intake.

By far the greater part of the section on treatment in each edition of his treatise on Diabetes is devoted to a consideration of the question how the carbohydrate intake can be reduced to a minimum or zero with the least possible inconvenience or danger to the patient, and gluten bread (first proposed by Bouchardat in Paris in 1841) and other substitutes for household bread are fully considered. But when we come to remedies we find the following statements in his first edition: "No medicinal agent, as far as I am aware, has yet been found that possesses the power of permanently diminishing the elimination of sugar in diabetes." He notes, however, the remarkable tolerance of opium by diabetics, and the temporary improvement under its use in two cases recorded by McGregor; and in the four cases in the appendix he had administered opium in one grain doses two or three times a day. But he does not expressly recommend it for the treatment of the disease, and on the last page, referring to "various remedies that have been proposed," he writes, "I have never seen anything recorded to give me an atom of faith in anything that has been proposed."

However, in his second edition he recommends alkalies, in the form of carbonate, or of their vegetable salts (citrate, acetate), and the alkaline mineral waters of Vichy, Vals, and Carlsbad; and he speaks highly from his own experience of opium in increasing doses. The writer remembers well the intense interest he took in a case which he was treating with full doses of opium, and his great gratification when he succeeded, not only in reducing the sugar to zero under dietetic treatment and opium, but in gradually withdrawing the opium and allowing an ordinary diet to be taken without the return of the glycosuria. Later, we know that he experimented upon the different alkaloids of opium, and, selecting codeia as the most suitable, prescribed it largely, a practice which was followed extensively by the profession in England.

Dr. Pavy's merits as a physiologist were well recognised in foreign countries, and many honours were conferred upon him. He was a corresponding member of the Anatomical Society of Paris, of the Medical and Chirurgical Society of Edinburgh, of the Society for "Innere Medicin" of Berlin, a Corresponding Fellow of the Pathological Society of Montreal, an honorary member of the Philadelphia Medical Society, and of the Royal Academy of Medicine, Belgium, an honorary Fellow of the Moscow Therapeutical Society, and a member (formerly a Vice-President) of the Paris Medical Society. In 1909 the Academy of Paris awarded him the Prix Godard for his work on Carbohydrate metabolism in diabetes.

Dr. Pavy had the defects of his qualities: with his tenacity and persistence in following out a line of thought there was a corresponding disinclination to look at the other side of the question. It was not always easy to discuss a case or a question with him; he appeared too convinced of the correctness of his own view to give anything like full consideration to other possible aspects. There were indications also that even within the range of his own subject he was not always fully alive to the work being done by other inquirers. This was excusable in one who pursued with such constancy independent research, probing deeper and

deeper into one of the most complex problems of physiology and pathology. His writings, if not marked by any literary distinction, had always the merit of being absolutely clear and unambiguous. If occasionally there was a suspicion of a cloud of words, or a lengthy phrase, or paraphrase, which might have been shorter, the cloud was never dense enough to obscure the light that was meant to reach the reader.

As already hinted, his views on the physiology of sugar were by no means widely accepted, and he was often engaged in defending his position against the attacks of his opponents.

Pavy has provided for himself a lasting memorial at Guy's Hospital. Through his liberality the authorities of the hospital were enabled in 1890 to construct a gymnasium within the precinct of the Residential College, known as the Pavy Gymnasium. The College had been built in the form of a quadrangle, and the piece of land allotted to it allowed of no more than was absolutely necessary for the ordinary College requirements, and a gymnasium was not then in question. The problem of introducing this luxury was solved by sinking it underground in the centre of the quadrangle, and lighting it by a glass roof, which projects in gable form a few feet above the level of the ground, with an adequate gangway round it. The gymnasium was formally opened in October, 1890. Dr. Pavy, till near the time of his death, had been its patron. Assaults-of-arms and gymnastic displays have from time to time been held, and Dr. Pavy was wont to preside at these meetings and present the prizes to successful competitors. Even in the end he did not forget the gymnasium, for he left in his will the sum of £2,000 as an endowment fund for its future maintenance.

In his home life Dr. Pavy was most methodical, and would have everything done in the way he wished. He liked to have the best of its kind, but he was not a follower of the fashion, and in his dress adhered consistently to the same mode for years in succession. He was particular as to the cuisine, and, as one should be who has written a book on Food and Dietetics, he was well known for his good dinners.

Reference has been made to his stylish equipage; and the writer remembers that Pavy was one of the first to have his house lit by the electric light, which before this method of illumination was installed all over London could be only got for domestic purposes from the Grosvenor Gallery. These were the days when a dinner party was frequently plunged into utter darkness by the sudden break in the supply; and this was perhaps not renewed until one or two hours had elapsed. Such an experience the writer well remembers at one of his entertainments.

Pavy was wrapped up in his work. He read general literature but little, and cared little for pictures; but was fond of music, and might be seen not infrequently at the opera. During the lifetime of his wife he often rode with her in Hyde Park, but afterwards he systematically took walking exercise, generally between afternoon tea and dinner, and if the weather was unfavourable he would drive to the Botanical Gardens and walk there under cover. For the most part he took his holidays abroad, but never left town until Parliament rose. He was fond of travelling, and for many years, as he told the writer, he made it his practice to visit the different spas and health resorts on the Continent in order to become fully acquainted with their advantages or disadvantages, with a view to advising any of his patients whom he might wish to send abroad. He went consistently to the several International Medical Congresses, including those held at Washington, Madrid, Lisbon, and the last at Budapest in 1909. He read papers at these congresses sometimes, and he was President of the National Committee of Great Britain and Ireland which worked in connection with them. He was one of a party of the British Association which visited South Africa in the winter of 1905. He was then 76 years of age, and went, as usual, unaccompanied by any of his relatives.

His eightieth birthday was the occasion for a wide expression among his professional friends of their appreciation of his distinguished career and of his eminent position as a physiologist.

At the next meeting of the Physiological Society, which took place at Oxford on June 26th, 1909, he was presented with a silver bowl bearing the inscription: "Frederick William Pavy, M.D., F.R.S., May 29th, 1909, from the Physiological Society in token of affection and admiration."

Dr. Pavy married, in 1855, Julia, the daughter of Mr. W. Oliver, and had two daughters. The younger died when about 17 years of age as they were returning from their summer holiday, having caught diphtheria in Paris. The elder daughter was married in 1881 to the Rev. Borradaile Savory, only son of Sir William Savory, Bart., the able surgeon to St. Bartholomew's Hospital. Three years later death stepped in again, and Pavy was left to mourn a handsome, accomplished, and devoted wife.

Pavy was a vigorous man, and knew very little illness in his own person. In the autumn of 1902 he had a sharp attack of pneumonia, but recovered completely. His previously erect form became somewhat bowed by age, but he retained his briskness and mental powers unimpaired. He maintained the routine of his daily habits, saw patients, continued his physiological researches, and was often at the Athenæum in the afternoon. In August, 1911, he went on the Continent for his summer holiday, and returned through Paris. Here first he seems to have become ill, and after spending a day or so in Folkestone, he arrived home on September 10th. Though very unwell, he managed to see patients, and did not seek any advice until September 15th. He then obviously had a rather extensive bronchitis, and though temporary improvement took place, he sank rapidly on September 19th.

There was a numerous attendance of his friends, colleagues, and other members of the profession at the memorial service held at St. George's, Hanover Square, and he was laid to rest in Highgate Cemetery.

A verbal description of one's hero, however detailed and accurate, still leaves an imperfect record; but in the present day it is usually easy to supplement the biography of any public character with portraits taken at different periods of his life.

The writer is not able to go farther back in Dr. Pavy's case than the time when he was a junior Assistant Physician. In the early sixties the members of the staff of Guy's Hospital were all separately photographed by Messrs. Maull & Polyblank, who took full-length portraits of *carte-de-visite* size. Dr. Pavy is seen as a rather slender figure of medium height, with a large head, broad high forehead, the hair somewhat scanty above, but in curly masses on either side; he has whiskers, but is otherwise clean shaven. He wears the wide-lapelled frock coat, the open double-breasted waistcoat, and the cravat tied in a large bow, which he wore until old age. He must have been then 33 or 34 years of age.

A larger photograph, probably taken about the same time, and representing him standing at a table with microscope, rabbit, and dissecting case in front of him, is in the possession of his family.

At the time of his retirement from Guy's in 1890, the *Guy's Hospital Gazette* reproduced a photograph by Jerrard. It must have been taken a few years previously, as it has the appearance of a man of not more than 52 or 53 years of age. It is a head and shoulders only, and the face is nearly profile. The head is still well covered with hair, which forms wavy masses at the back, the thin lips are firmly compressed, and the face wears a slight smile of satisfaction and contentment.

The portrait which accompanies this memoir is a photogravure reproduction by the Swan Electric Engraving Co. of an oil painting by Percy Bigland. The picture hangs on the wall of the staircase leading to the Court Room of Guy's Hospital, and represents him shortly after his retirement from the active Staff. It is a sitting figure, with left three-quarter face. There is less hair than formerly, and it is less bushy behind; the expression is the same as in the preceding, and the costume, with the wide expanse of shirt front, and the black bow are the same.

The obituary notice of Dr. Pavy in the *Lancet* was accompanied by a reproduction of a portrait by Messrs. Maull & Fox, of 187, Piccadilly, who have been good enough to inform

the writer that the photograph was taken in August, 1908, when Dr. Pavy was 79 years of age. It is a full-faced portrait, and the difference in years since the Guy's Hospital painting is unmistakable.

Lastly may be mentioned the drawing in crayon by Mr. W. Strang, A.R.A., which was made in 1908, and is in the possession of the Royal Society of Medicine. Many of his old friends and admirers have *fac simile* copies of this excellent work, and a smaller copy of it illustrated the obituary notice of him in the *British Medical Journal*. One notices the less upright carriage of the head, the deeper lines of the face, and the somewhat strained appearance of the features which are common in those of advanced years. Dr. Pavy was then about 79 years old, and the shortening of stature and bowing of the figure, so characteristic of the end of the eighth decade, had been noticeable in him for some time.

FREDERICK TAYLOR.





# CASES OF POISONING BY OPIUM.

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By

FREDERICK TAYLOR, M.D.

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IN April, 1912, I read a paper before the Therapeutical and Pharmacological Section of the Royal Society of Medicine in which I advocated the more frequent use of the Faradic current for the treatment of the severer cases of poisoning by opium.\* In pursuit of my argument I thought it desirable to examine the records of cases of opium poisoning at Guy's Hospital; and it may interest the readers of these reports if I give them the results of this research in an analysis and brief accounts of the cases recorded.

In the fourteen years 1898 to 1911, inclusive, there were 55 cases coming under the head of acute poisoning by opium or morphia. Cases of morphiomania are not included. The 55 cases were of all degrees of severity; in some the symptoms were so mild as to make one doubtful if any poison was really taken; on the other hand, three cases were fatal.

All but three of these cases were treated in the Clinical wards; and I should say for the information of those unconnected with Guy's Hospital that the Clinical wards are wards set apart for the reception of cases which are especially likely to be of service for the preparation of clinical lectures; that they are under the charge of all the Physicians and Assistant Physicians in turn in the course of the year; and that they are constantly served by a staff of senior students in their fourth or fifth year, many

\* The Treatment of Opium Poisoning by the Faradic Current. Proc. of the Roy. Soc. of Med., May, 1912. Therapeutical and Pharmacological Section, p. 165.

of whom are already qualified to practise. I am indebted to my colleagues for permission to publish these cases.

*Sex.*—Of the 55 cases, 30 were males, 24 were females, and in one case, a child, the sex is not mentioned.

*Age.*—The ages vary from 3 months to 73 years. The decades most frequently represented are the third, fourth, and fifth. Thus, 9 cases occurred with ages between 20 and 29, 12 cases with ages between 30 and 39, and 12 cases with ages between 40 and 49. All in the first decade were under 5 years of age.

*Form of the Poison.*—This is best shown in the following table :—

Laudanum or tincture of opium	...	...	...	34
Solid opium or powder	...	...	...	3
Medicine containing opium...	...	...	...	2
Syrup of poppies	...	...	...	1
Lead and opium lotion	...	...	...	5
Nepenthe	...	...	...	1
Chlorodyne	...	...	...	3
Horse medicine	...	...	...	1
Morphia by injection	...	...	...	1
Form unknown	...	...	...	4

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55

*Quantity.*—The amount of laudanum varied from a drachm to two ounces, or “a lot.”

One drachm	was the amount taken in	1 case.
Two drachms	“ “ “	5 cases.
Three	“ “ “	1 case.
Four	“ “ “	3 cases.
Five	“ “ “	2 cases.
One ounce	“ “ “	4 cases.
One and a half ounces	“ “	1 case.
Two ounces	“ “	2 cases.
One pennyworth	“ “	1 case.
Two	“ “	5 cases.
Three	“ “	2 cases.
Four	“ “	2 cases.
“A lot”	was taken in	2 cases.
The quantity	was not stated in	3 cases.

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34 cases.

The quantity of opium powder was 40 grains in one case and a drachm in another; and a baby aged six months had a "soothing powder." The medicines were cough mixtures containing compound tincture of camphor. Three drachms of syrup of poppies were taken by a child aged 18 months. The lead and opium lotion of the Guy's Hospital Pharmacopœia contains a mixture in equal parts of lotio opii and lotio plumbi (liquor plumbi sub-acetatis dilutus, B.P.), and contains  $2\frac{1}{2}$  grains of extract of opium in each ounce. The quantities taken were, in two cases, one ounce, in one case half a pint, and in two cases "half a bottle." Probably the bottle was in each case the wine bottle frequently employed to take away a week's medicine, but there is no information on this point. It is, of course, difficult to know how much opium was really taken. In three of these cases the symptoms were mild, in one severe, and in one quite serious. The quantities of chlorodyne taken were one ounce in one case, and half an ounce in two cases.

In at least four cases there was evidence of alcohol having been taken freely before the opium; in one case the laudanum was supplemented by one pennyworth of paregoric; and in another case by two drachms of nepenthe.

I have inquired of chemists what a pennyworth of laudanum represents. Two have told me that half a drachm or a drachm of tincture of opium is often given for one penny, two drachms for twopence, or as much as an ounce for fourpence. A third stated that he would avoid, as far as possible, selling definite quantities of laudanum, but would give a few drops only in some excipient, in order to deal with the ailment which the purchaser put forward as the reason for his wanting it.

*Symptoms.*—Probably there is little or nothing to be added to existing knowledge on this subject from a consideration of these reports. As an experiment on the effects of opium poisoning, the ordinary case of the person accidentally poisoned or taking the drug with suicidal intent has little value. The quantity taken is often unknown, the time at which it is taken is uncertain, the effects are sometimes complicated by alcohol

previously ingested, and frequently either immediately or within half an hour or an hour the observation is spoilt by the use of an emetic, the operation of the stomach tube, or the administration of an antidote. One patient walked into the Thames after taking two drachms of laudanum, and added the collapsing influence of cold to that of the poison. The only exceptions to the rule that drowsiness, sleepiness, heavy sleep, and coma were the progressive results of the poison were in a few cases in which the dose was very small (Cases 16 and 46), or in which emetics or lavage had been so promptly used as to prevent any material absorption (Cases 18, 20, and 49). But in most cases it is so difficult to consider the symptoms apart from the treatment that it seems desirable that some account of the treatment employed should be given at once.

The evacuation of the stomach was, of course, the first consideration, and this was done in the majority of cases by means of the stomach tube, but in a few cases, especially three which were dealt with first by the police, an emetic was given.

In five cases only is there no note of the stomach being cleared. They were cases in which the symptoms were not very severe: one was a baby (Case 46); another a child set.  $3\frac{1}{2}$  (Case 2). In 18 cases an emetic was given. In six of these the emetic failed to act, and five of them were washed out. In the remaining 12 cases vomiting took place, but six of these were subsequently washed out. Four patients vomited spontaneously before coming under treatment, and these were all washed out. Altogether, the stomach was washed out in 43 cases. In 31 of these a solution of potassium permanganate was introduced as an antidote; in the remaining 14 cases there is no mention of potassium permanganate, though it is possible that in six of these (Cases 13, 15, 17, 25, 43, and 51) potassium permanganate was used, for at that time the treatment by this means was so frequently employed that the statement that the patient was "washed out" may have been considered to be a sufficient account. In the other eight cases it is either specifically stated that the patient was washed out with water,

and that he was washed out at the police station; and I assume (perhaps wrongly) that there the fluid used would have been water only. The analysis of the 31 cases in which potassium permanganate was used shows considerable variety in the strength of the solution.

The Guy's Hospital Pharmacopœia contains a *lotio potassii permanganatis* of 1 per cent. strength. In 12 cases this was diluted with from four to nine parts of water, so that the strength would have been from .1 to .2 per cent.; in five cases the fluid used was a "weak solution," a "dilute solution," or "water tinted with" or "containing" the permanganate. In seven cases a solution of potassium permanganate is mentioned as having been employed, without further description. One patient was washed out with "5 pints of permanganate of potassium solution of 20 per cent. strength." I take this to mean that the solution contained 20 per cent., or a fifth part of the liquor, not of the solid salt, and that, therefore, it contained really .2 per cent., and not 20 per cent. In this case, also, pills containing a quarter of a grain of the salt were given every four hours, but there is no record of the number taken.

In two cases (20 and 21) a 1 per cent. solution of permanganate is said to have been used, but as this is the strength of the official liquor *potassii permanganatis*, and two and a half times the strength of undiluted Condry's fluid, it is probable that this is a mistake in reporting. The quantity used for lavage varied from one to five pints, and in a few cases the washing was repeated after an hour or half an hour. In a few cases, also (six at least), it is stated that some of the solution was left in the stomach, the amount varying from three to ten ounces.

The late Dr. Murrell recommended the use of 10 grains of potassium permanganate in 1 pint of water, which is equivalent to a .1 per cent. solution, and this, he says, should be repeated in half an hour.

The Guy's Hospital Pharmacopœia contains a section on "Poisons, Antidotes, and Treatment" for the guidance of House

officers and others dealing with patients admitted to the hospital. The only antidotes there recommended in the edition of 1899 are potassium permanganate, and atropine or belladonna. The directions with regard to the former are as follows:—"Wash out stomach at half-hour intervals with solution of potassium permanganate, diluted with five times the quantity of warm water, leaving about 5 ounces of the diluted solution in the stomach." Since the Guy's Hospital solution of potassium permanganate has a strength of 1 per cent., the solution to be employed has a strength of .16 per cent. If 5 pints are employed for irrigation of the stomach, the amount of permanganate put into the organ is about 80 grains, and the quantity of fluid ordered to be left in should contain about 4 grains of the salt.

The preceding edition, published in 1891, has no reference to potassium permanganate, and it is in this interval between 1891 and 1898 that attention seems to have been directed to its possible value in cases of poisoning by opium. The fact that morphia decolourised a solution of potassium permanganate, in definite relation to the quantities of the two substances, was applied by Dr. Barker Smith as long ago as 1877 to the quantitative estimation of the alkaloid in solution. He found practically that 5 milligrammes of potassium permanganate were decolourised by 4 milligrammes of morphia.

The process is one of oxidation, forming oxydimorphine (Kobert), and the extension of this principle to the use of potassium permanganate in poisoning by opium was made in some cases reported by Dr. Moor in the Medical Record of New York, March 2nd, 1895. In a case of profound coma from opium poisoning one grain of potassium permanganate dissolved in 10 minims of water was injected, and later another grain; ten minutes after the last a considerable improvement in the symptoms took place. In another case seven grains of morphia had been taken. As an antidote 10 grains of potassium permanganate in a tumblerful of water were given to the patient. Then, an hour and a quarter after the morphia had been taken three grains of the salt in two drachms of water were injected

subcutaneously, and this was repeated twice at intervals of 15 minutes. After the last injection there were signs of returning consciousness. In another case  $3\frac{1}{2}$  grains of the salt were injected subcutaneously; half an hour later 16 grains in solution were put into the stomach, and half an hour later another injection of  $3\frac{1}{2}$  grains was made. Soon after this there were signs of returning consciousness.

In the same journal, page 648, a case is recorded in which an ounce and a half of laudanum had been taken. The treatment was subcutaneous injection of one grain of potassium permanganate at half-hourly intervals until three grains had been given; two hours later three grains were injected into the buttock; an hour later three grains more. The patient's condition, nevertheless, was worse an hour later, when again three grains were injected. Half an hour later the respirations were only 6 in the minute; but at 2 o'clock they were 10; at 2.30 they were twelve, and ultimately recovery took place.

Belladonna or atropine was also used as an antidote in 18 cases only, or one-third of the number. A child, aged  $3\frac{1}{2}$  years, had 8 minims of tincture of belladonna, and an infant, aged 3 months, had half a drachm of *mistura belladonnæ composita*, containing half a minim of tincture of belladonna. Twelve cases had 3 minims of *liquor atropinæ* injected subcutaneously, and in four of these the dose was repeated. In two cases 5 minims were injected, and in one of these a dose of 4 minims was given afterwards. In another case a man had a dose of 2 minims only, and a man aged 63 had 1 minim only, followed by a second dose of the same strength.

Hot coffee by the mouth, or coffee by enema, was given in 18 cases; and in a few cases ammonia, amyl nitrite, ether by injection, or strychnine by injection, was administered. In three cases only was Faradism employed, and these were the worst cases, that is, those which were fatal (Cases 13, 14, and 37).

To return to the symptoms observed, the only exceptions to the rule that drowsiness, sleepiness, heavy sleep, and coma were the



progressive results of the poison, are a few cases, in which either the dose was very small (Cases 16 and 46), or emetics or lavage had been so promptly used as to prevent any material absorption (Cases 18, 20, and 49).

In many cases it is difficult to say that the treatment by emetics, lavage, with or without the use of potassium permanganate had any effect. The patients continued drowsy or sleepy and were kept from falling into a hopeless state of coma either by ambulation or by rousing while in bed, and this mechanical treatment had to be continued in some cases for several hours, or a whole night, before the patient was so wakeful, or, if sleepy, so easily roused, as to be thought free from further risk.

In some cases, however, the symptoms appeared to be quickly modified by the process of lavage, but whether this was due to lapse of time or to the stimulation of the surface involved in stomach tube operations, or to the action of potassium permanganate when it was employed, it is difficult to say. Case 7 was admitted comatose with pin-point pupils, cold extremities, his breath smelling of opium, the pulse 70, and respirations 12. He had 3 minims of atropine solution injected, and was washed out in the surgery, but no mention is made of potassium permanganate. The washings were brown and smelt of opium. "The comatose condition rapidly passed off," and when removed to the ward he was so violent that the attempt to administer a coffee enema had to be abandoned.

Case 8 was much improved after the second lavage, but she had vomited, had had coffee, and had been walked about freely besides.

Cases 17 and 42 are similarly reported; and Case 52 was better soon after an injection of ether.

On the other hand, in some cases the patients were worse after lavage, not, we may suppose, because of the lavage, but because the poison already absorbed was then beginning to take effect.

Case 11 was washed out with .11 per cent. solution of permanganate of potassium, was injected with atropine, and had a coffee enema. After the atropine the pupils dilated; after the coffee the patient lost consciousness, but was easily roused by ambulation.

Case 39, after two washings with potassium permanganate solution, of which 10 ounces were left in the stomach, became drowsy, and increasingly so, so that further measures were considered necessary, including the attentions of a nurse to keep the patient awake.

Case 50, after having been watched for two or three hours, sent out apparently as safe, dragged back by the police, washed out with potassium permanganate and injected with atropine, was still sleepy and difficult to arouse.

Case 23 was that of a child, aged 4, to which an overdose of medicine was given. It was immediately discovered, and mustard was given so that it vomited; it was brought to the hospital, and was washed out with potassium permanganate. After this it got drowsy and the pupils contracted, but on being put to bed it cried loudly. It is quite likely here that the drowsiness was a natural sleep, and that what little opium had been taken was either entirely rejected or neutralised sufficiently to be harmless.

Of the effects of atropine it is also difficult to speak with any certainty in most cases. The drug was often given in combination with the use of potassium permanganate, in most case it was only given once; it was preceded or followed by mechanical methods of rousing the patient. Nor would one expect to see a sudden cessation of narcotic symptoms either immediately or within a short period of its administration. Nevertheless, out of the 18 in which it was given, there are 4 cases in which its own poisonous effects appear to have manifested themselves. In Case 29, after admission to the ward, a dose of atropine, equivalent to 1/20 grain, was administered. Two days later the patient's condition is reported as satisfactory, and the pupils were widely dilated; in the evening he was very lively, trying to get out of bed, and using unparliamentary

language. The following morning he presented an erythematous or scarlatiniform rash, which was fading two days later.

Case 32, admitted drowsy at 4 p.m., and injected with liquor atropinæ, 3 minims ( $1/33$  gr.), and wanting constant attention to keep him awake, became less drowsy; he was suspicious and excited, and his pupils dilated. At 9 p.m. he tried to get out of bed, shouted and struggled. He was noisy for half an hour, then slept, but was noisy when waked, and had to be removed to the strong room at 11 p.m.

Case 55 had the same dose ( $1/33$  gr.) in the surgery, and was led to the ward. Here he talked cheerily and incessantly, but was seized by an overpowering wish to sleep at times. The following day the symptoms of opium poisoning had disappeared, but he had some headache and "occasional fits of shaking," which were attributed to resecreted morphine. These appeared to be partially checked by irrigation of the colon.

In three cases only is it recorded that electrical treatment was employed. These were all fatal, and it is clear that recourse to this method was only had when the patients were in an extremely grave condition. It is disappointing to me to find that so little good result was obtained from it, since in my private experience, as already mentioned, I have had to do with two cases in which it appeared to me that the Faradic current was of the greatest value, and largely contributed to the recovery of the patient. The Guy's Hospital cases are Cases 13, 14, and 37. I have so fully discussed this subject in the paper previously mentioned,\* which was based on the above two cases, that I need not enlarge upon it here.

Authors are not quite uniform in their recommendations as to the use of the Faradic current. Some regard it as useless, or even likely to do harm by exhausting the patient. Others recommend it rather as a *dernier ressort*; some would apply it by a wire brush electrode to stimulate the sensory nerves; others apply

\* The Treatment of Opium Poisoning by the Faradic Current. Proc. of the Roy. Soc. of Med., May, 1912. Therapeutical and Pharmacological Section, p. 165.

it indiscriminately to the surface without specifying the nature of the electrodes; others only suggest that the phrenics should be faradised when the respiration is failing, and this in combination with artificial respiration. Books I have consulted on electrotherapeutics do not deal specifically with its use in opium poisoning.

In the Guy's Hospital Pharmacopœia, both in the edition of 1891 and in that of 1899, "Faradism" is included among remedies, without any indication as to how, when, or where the Faradic current should be applied.

From the reports of the cases to which I have referred it is clear that it was applied as a general stimulant to the surface of the body and limbs generally. In Cases 13 and 14 it appears to have had no result whatever; in Case 37 the record is that the breathing was improved, and that reflexes returned under its influence; it was then stopped; the conditions again deteriorated; they were a second time improved by Faradism, with oxygen inhalations and ether injections; but ultimately death took place.

Of special features of opium poisoning we may note the conditions of the pupils, of the pulse, and of the respiration. The pupils are described as pin-point pupils in 7 cases; as minute, or extremely contracted, in 3; very small in 4; small, or contracted, in 20; not very contracted, 2; moderate or medium, 4; not contracted, 2; dilated, 1; not dilated, 1; no mention of their condition, 10; total, 55.

The pulse was very variable. The slowest recorded pulse was in Case 19; it was counted in different stages at 60, 65, and 80, with respirations respectively 8, 12, and 20. But this patient, after having taken 2 drachms of laudanum, had walked into the Thames, and was admitted in a collapsed condition, obviously in part attributable to loss of heat from immersion.

Case 13, which was fatal, had a slow, irregular pulse; Case 11, a slow, weak pulse; the pulse in Case 7 was 70; but no other case is recorded to have had a pulse slower than 80, and some pulses were as high as 120 and even 140.

The number of cases in which the respirations are recorded below 16 is also small. In Case 3 the respirations were at first 8, then 14, and 16; in the fatal case, 13, they were 9, and irregular; in Case 19, already mentioned, they were 8, 12; and in the fatal case, 14, though they were no less than 16 in number per minute, they had, during the last stages, the Cheyne-Stokes' character. On both of these points, however, the records are defective.

The most serious cases were Cases 3, 7, 13, 14, 34, 37, 39, 50, and 53. Of these, three were fatal, Cases 13, 14, and 37.

## REPORT OF CASES.

CASE 1.—1898, F. æt. 45.—July 23rd: Took two drachms of laudanum; told her husband who came up to the hospital. The senior obstetric resident went to her home; found her comatose; washed out her stomach; gave her liquor strychninæ; and brought her up to the hospital. C.O.A.: Sleepy, but easily roused; pulse and respiration normal; pupils of medium size. She was seven months pregnant. The stomach was washed out with water tinted with potassium permanganate, and half a pint was left in, but this she vomited. Thirty minims of ether were injected hypodermically. She showed no further tendency to go to sleep, and was awake all night. July 24th: The urine gave a morphia reaction; patient practically well. July 26th: Went out.

CASE 2.—1898, F. æt. 3½ years.—September 9th: In the afternoon drank a cupful of *mistura ipecacuanhæ* co. (containing 4 minims of *tinctura camphoræ* co. in one fluid drachm). There was no vomiting, but the child was drowsy for three hours afterwards. C.O.A.: Very drowsy, but could be roused with no great difficulty. Pupils were not dilated (? contracted). Respirations quick; skin hot and dry; no smell of opium in the breath. She was put in a hot bath and afterwards to bed, and 8 minims of *tinctura belladonnæ* were given at 11.30 p.m. She passed a good night and seemed to be none the worse the following morning. The amount of opium taken was calculated at the time to be half a grain.

CASE 3.—1898, M. æt. 39.—November 12th: Was thought to have taken an ounce of laudanum about 11.30 p.m. In less than an hour he was comatose. At 1.30 a.m. the stomach was washed out; at 2.30 it was again washed out, this time with potassium permanganate, and hot coffee was introduced into the stomach. At three o'clock the heart was feeble, the respirations 8 per minute; the pupils pin-point. Atropine, 1/20 gr., was injected at 3.15; strychnine, 1/20 gr., at 3.30; and artificial respiration was applied for three-quarters of an hour from 3.30 onwards. The pulse and breathing were then better, and he was brought up to the hospital. C.O.A.: 4.30 a.m., unconscious, extremities cold, skin moist; pulse, 120; respirations, 16, stertorous. If the tongue is allowed to fall back breathing stops entirely. Reflexes are absent. Pupils, 2 mm. in diameter. Hot bottles were applied. At 6 a.m. the pulse was 130; respirations, 14; and the pupils were 3 mm. in diameter. 7 a.m.: Pulse, 124; respirations, 14; rather irregular; pupils, 3½ mm.; slight conjunctival reflex; hands warmer; atropine, 1/25 gr., injected. 8 a.m.: Pulse, 108; respirations, 14; pupils the same size;

slight reaction to light; conjunctival reflex good; a pint of strong coffee injected into rectum. At 9 a.m. tendon reflexes were present; and at 10.30: Pulse, 132; respirations, 12, irregular; partially conscious; pupils 4 mm. 11.30: Can now understand what is said to him, and tries to answer simple questions. At 12.30 he was dozing quietly, and at 2.30 p.m. was quite conscious. The following day, November 13th, he was well; pulse, 88; respirations, 20; pupils, 5 mm. November 14th: A trace of albumen in the urine. November 15th: Albumen absent; went out.

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CASE 4.—1899, F. æt. 8 months.—May 16th: The mother gave it half a drachm of cough mixture containing paregoric. After that it became drowsy and refused the breast. The mother then gave it 20 minims of vinum ipecacuanhæ, but no vomiting took place. C.O.A.: Drowsy, cyanosed; breathing slowly, and stopping every now and then; pupils moderately contracted; pulse good; slight physical signs of bronchitis. It was put in a cot with hot blankets and hot bottles. Twenty minims of vinum ipecacuanhæ with hot water and tickling the throat with a feather produced only slight sickness. Later (12.30) the stomach was washed out with hot water; the breathing became better almost at once, though the child was still drowsy. During the afternoon a drachm of mistura belladonnæ composita (containing 2 minims of tinctura belladonnæ in the drachm) was ordered every two hours. Towards the evening the baby took food, the pupils enlarged, and respirations were more natural. May 17th: Went out.

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CASE 5.—1899, F. æt. 37.—Has often tried to commit suicide. July 19th: Went into a beershop and said she had taken opium. Was given some salt and water, and sent to the hospital. C.O.A.: Drunk; does not appear to be under the influence of opium, though the breath smells of it; was washed out at once with several pints of warm water and put to bed. Half a pint of coffee was given. She did not get any worse, and was discharged July 22nd.

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CASE 6.—1899, F. æt. 41.—Supposed to have taken two drachms of laudanum, but the time is not known. At 8 p.m., July 21st, brought up by the police. In the surgery, comatose; no response; pupils small, but not quite pin-point; no reaction to light; good colour; good pulse; respirations, 18, regular. She struggled when the stomach tube was passed, but was washed out with 4 pints of water. After this she was still drowsy, and the pulse was rather feeble. Taken into the ward; some hot coffee given by the mouth. At 10 p.m. she was sleeping quietly. July 22nd: Some headache; otherwise well. July 23rd: Went out.

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CASE 7.—1900, M. æt. 48.—On December 30th, 1899, he felt suicidal, and told an undertaker, a relative of his, to come round and measure

him for his coffin. The undertaker then found that he had taken a quantity of laudanum, and sent for the police, who brought the patient to the hospital. C.O.A.: Comatose, with pin-point pupils: extremities cold; breath smells of opium; pulse, 70; respirations, 12; atropine, 3 minims, injected; stomach washed out in the surgery, the brown washings smelling of opium. Then the comatose condition rapidly passed off. He was taken into the ward, and when an attempt was made to give him a coffee enema he became so violent that it had to be stopped. He was left alone and he gradually improved, the pulse getting stronger, limbs warm, and the pupils, by night time, almost normal. December 31st: Some headache, otherwise well. January 1st, 1900: Well. January 2nd: Went out.

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CASE 8.—1901, F. æt. 26.—At 6 p.m. on January 2nd, took a wine-glassful of laudanum, and went to bed. At 7 p.m. felt drowsy and called her neighbours, who prevented her from sleeping. At 10 p.m. a doctor gave her an emetic; she was sick. This was followed by potassium permanganate, the quantity of which is unknown, and she was walked about for an hour, and then brought to the hospital. C.O.A.: 11.30 p.m., drowsy and apathetic, but conscious and could walk with support; no reflexes present; pupils minutely contracted; pulse rapid, feeble. Washed out twice with solution of potassium permanganate 2 per cent. with an interval of half an hour, 5 ounces of solution being left in; ether, 60 minims, injected subcutaneously. By the end of the second lavage she was warm, sensible, no longer so drowsy; pulse, 72; respirations, 24; reflexes normal. Slept indifferently after this. January 3rd: Feels now well; went out.

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CASE 9.—1901, F. æt. 48.—Said to have taken two pennyworth of laudanum six hours previously. C.O.A.: Pulse, 88; pupils a little contracted; retching occasionally. An ounce of ammonia mixture and a coffee enema, one pint, were administered. She slept comfortably all night, and went out the next morning.

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CASE 10.—1902, M. æt. 28.—On December 4th, between 10 and 11 p.m., while intoxicated, took half an ounce of chlorodyne; was washed out and had an emetic in the surgery, but did not vomit. C.O.A.: Depressed, though perfectly conscious; quite quiet; took milk and hot coffee during the night. Another statement was to the effect that he was washed out with solution of potassium permanganate, and that traces of morphia were found in the urine. He went out well December 6th.

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CASE 11.—1902, F. æt. 53.—On March 15th took two pennyworth of tinctura opii, and soon became drowsy. Had to be supported



to the surgery ; was sleepy, pale, cold, with slow, feeble pulse, but no material disturbance of breathing. Washed out with solution of potassium permanganate of .11 strength. Liquor atropinæ sulphatis, 3 minims, injected. Half a pint of hot coffee was ordered to be given during the night. The pupils enlarged after the atropine, but soon after taking the coffee she lost consciousness, though easily roused again; and she was walked up and down in the open ten minutes, and was thus effectually roused. About 5 a.m. the pupils were still very small. The dose of atropine was repeated. She went to bed a few hours later, "after breakfast," and slept normally until the afternoon. She went out on March 17th.

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CASE 12.—1902, F. æt. 33.—On May 26th, about midnight, took some laudanum. A doctor gave her some mustard and water, but she was not sick. At 12.50 in the surgery she was stupid, but not sleepy ; pupils normal ; pulse and respiration normal. Was washed out with solution of potassium permanganate of .2 per cent. strength, and some was left in. Pulse, 100 ; respirations, 24. Coffee was also given. She was wakeful all night. In the morning she had some headache. Went out May 27th.

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CASE 13.—1902, M. æt. 24.—The history is obscure. He appears to have had some fits. He and some friends had been drinking, and he fell in the street about 2 a.m. on August 9th, and was seen by a doctor who injected something into his arm. In the surgery at 4 a.m. he was comatose, with cold skin, contracted pupils, blue lips, pulse slow and very irregular ; respirations, 9, irregular ; limbs lax ; corneal reflex sluggish. He rallied somewhat from his collapsed condition with warmth and strychnine injection, but there was no certain information as to his having taken a narcotic. However, his stomach was washed out, and a Faradic current was applied vigorously about 5.30 a.m. for about 20 minutes. Probably it was used again at intervals, but he remained in much the same condition, and died about 2 p.m. The stomach washings smelt of alcohol. Later the stomach contents were found to contain opium. A post-mortem examination showed the organs to be healthy.

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CASE 14.—1902, M. æt. 63.—On August 13th, about an hour before admission, he was found unconscious, with a two-ounce bottle in his pocket, containing a few drops of laudanum. At 7.15 p.m. in the surgery he was unconscious, could not be roused ; breathing 16 in the minute, irregularly, stertorously, and blowing the cheeks out ; limbs quite lax ; no sensation ; pupils very small, not reacting to light, though the corneal reflex was good ; pulse full, regular, 80. He was washed out with water and then with a potassium permanganate solution, and a strong Faradic current was applied to the face, trunk, and limbs. He made no sign. He was then removed to the wards, where at 8.15 he was

still comatose, with cyanosed lips and cheeks, and dilated venules. The skin was dry and warm; the pulse regular. The Faradic current was again applied more than once. At 9 p.m. the pulse began to fail. The breathing took on the Cheyne-Stokes' character, and was unaffected by amyl nitrite. He became more cyanosed, profoundly comatose, artificial respiration had no effect, and he died at 12.30. Inhalation of oxygen, strychnia injections, and coffee enemas were also employed.

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CASE 15.—1902, F. æt. 19.—September 25th: Took two tablespoonfuls of some poison, probably laudanum; half an hour later came to the surgery. She was slightly drowsy, but the pupils were not contracted. Washed out; three grains of calomel were given; she was made to walk about the ward for an hour or two; ether, 10 minims, subcutaneously; a cup of black coffee. September 26th: Quite well; went out.

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CASE 16.—1902, M. æt. 69.—November 11th: Took half an ounce of chlorodyne at 9.45 a.m. Within half an hour the stomach was washed out with a weak solution of potassium permanganate. C.O.A.: Described as silly; pulse and respiration healthy; pupils reacting to light and accommodation; appears to have had few or no symptoms of opium poisoning.

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CASE 17.—1902, F. æt. 57.—December 13th: In the afternoon took one pennyworth of laudanum, and was found lying on a bed somewhat drowsy. Was made to get up, and walked unsteadily downstairs; shortly afterwards became unconscious. In the surgery she was unconscious, with minutely contracted pupils. Ether, 20 minims, subcutaneously; and she was washed out. She became more conscious; then had *mistura ammoniæ*, 2 ounces, and was taken to the ward, where one pint of coffee was given per rectum. Shortly after this she was got on to her feet, and could walk a little. Kept awake for some hours by being made to walk up and down the ward; pulse good; pupils less contracted. December 14th: About 1 a.m. put to bed; brandy  $\frac{1}{2}$  ounce every six hours; later, headache; otherwise well. December 15th: Quite well; went out.

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CASE 18.—1903, F. æt. 42.—May 2nd: Took two drachms of laudanum; was found by her son 15 minutes afterwards; 10 minutes later the police surgeon gave her an emetic, and she vomited. In the surgery she was perfectly conscious, and answered questions intelligently; pulse, 118; respirations, 16; pupils small, but reacting to light. She had some hot coffee; was walked about half hourly during the night. May 4th: Went out well.

CASE 19.—1903, F. æt. 42.—September 28th: Appears to have taken 2 drachms of tinctura opii, and then walked into the Thames at Southwark bridge. At 12 noon in the surgery: wet, collapsed. Hypodermic injection of strychnine, 2 minims. In the ward she was placed in hot blankets, and a mixture of liquor ammoniæ and infusion of digitalis was given. She was washed out with 4 pints of solution of potassium permanganate of .14 per cent. strength. During the afternoon she improved; she was very drowsy, but could be roused easily; pulse, 65; respirations, 12. September 29th: Better; pulse, 80; respirations, 20. October 1st: Went out.

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CASE 20.—1903, F. æt. 38.—November 7th: Appears to have taken half an ounce of laudanum. Found by a constable within 15 minutes. Cold. Pulse, 72, fair; pupils reacting to light, not very contracted. Washed out with a 1 per cent. solution\* of potassium permanganate; sent into the ward. Soon better. November 8th: Went out.

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CASE 21.—1903, M. æt. 21.—November 8th: Drank an ounce of lead and opium lotion. On arrival at Guy's was walked up and down the colonnade by the police for about an hour. C.O.A.: In the ward somewhat drowsy, hands and feet cold; pupils moderately contracted, react to light; pulse slow and full. The record on the chart is—pulse, 80 to 88; respirations, 24 to 26; temperature, 98°—97.2°. Stomach washed out with a 1 per cent. solution\* of potassium permanganate; put to bed; hot-water bottles. Instruction—to be waked every ten minutes. November 9th: Went out.

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CASE 22.—1903, F. æt. 30.—November 17th: Took half an ounce of tincture of opium and one pennyworth of paregoric. Came to the hospital within an hour; dazed, but answered questions; pupils somewhat contracted, react to light; temperature, 97°; pulse, 100. Washed out with five pints of water, then with five pints of permanganate of potassium solution of 20 per cent. strength.\* Admitted to ward, and was ordered calomel, 4 grains, and potassium permanganate pills, containing a quarter of a grain, every 4 hours. Pupils less contracted; wide awake, though somewhat dazed; nurse not to allow her to sleep during the night. November 18th: Head aching, otherwise she is well; went out in the afternoon.

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CASE 23.—1903, Child, æt. 4.—A drachm of tincture of opium was given by mistake. This was discovered immediately, and vomiting was caused by mustard and water. Stomach washed out with 2 pints of solution of potassium permanganate of .1 per cent. strength, and 3 or

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\* See page 29.

4 ounces were left in. In the surgery the child became drowsy, and the pupils contracted. It was put to bed, and then cried loudly. It again got drowsy, and the pupils contracted. No lividity. Coffee was given by mouth and rectum, and by midnight the pupils were large. At 2 a.m. the child woke, and made a lot of noise. It was allowed to sleep after this; went out well.

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CASE 24.—1904, M. æt. 48.—Took half a bottleful of lead and opium lotion at 2 a.m. on January 12th. At 10.20 a.m. had not been sick; not now drowsy; sweating freely; pulse, 80, small; respirations shallow; pupils small. Zinc sulphate, half a drachm; vomited fluid with no odour of opium. In the ward washed out with solution of potassium permanganate of .11 per cent. strength. At 1 p.m. *mistura magnesiae cum magnesiæ sulphate*, two ounces. Drowsy during the afternoon, but easily roused; pupils smaller than in the morning; no collapse; bowels moved at 4 p.m. No further signs of poisoning. January 13th: Went out.

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CASE 25.—1904, M. æt. 26.—January 27th: At 8 p.m. he left his brother's house, being in a state of depression. At 11 p.m. the police found him in his rooms sitting on his bed. He resisted being moved. At the police station an emetic was given without result. C.O.A.: Violent, struggling, rigid, grinding his teeth, smelling of alcohol; pupils contracted; pain in the cardiac region. Washed out in surgery. Drowsy, but could easily be roused; and then talked rationally. Some strong coffee was given, but was soon vomited. "He continued drowsy, but his friends and the policemen spent the night keeping him awake." January 28th: Headache; professes to know nothing of last night's events. January 29th: Went out.

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CASE 26.—1904, M. æt. 22.—March 7th: Brought up by the police, supposed to have taken four pennyworth of laudanum; he thought about 200 drops. Slept on the way to the hospital; was drowsy; pupils not contracted. Well washed out with solution of potassium permanganate in the surgery. He walked to the ward, was not put to bed, but kept up till 10 p.m. Ammonia mixture was given to him. He was drowsy and depressed; the pupils became smaller, but never very small; he took food as well as some hot coffee, and was disposed to talk rationally. When in bed remained awake for some time; ultimately slept well. March 8th: Went out well.

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CASE 27.—1904, M. æt. 35.—Suffered from pain in the knee, and often took 20 grains of *pulvis opii* for this. On May 29th he took 40 grains, and, feeling unusually drowsy, he came to the hospital.

He was drowsy, with cold skin, feeble pulse, small pupils; pulse, 80; respirations, 16; temperature, 98.4°. He vomited after a dose of zinc sulphate, and the vomit smelt of opium. He drank 3 pints of solution of potassium permanganate of .16 per cent. strength, and, after stimulating the pharynx vomited freely again. Hot strong coffee was given to him at intervals. March 30th: Well, but had not passed urine, and the catheter was used. March 31st: Went out.

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CASE 28.—1904, F. æt. 16.—July 2nd: In a fit of temper swallowed an ounce of lead and opium lotion. A policeman was called in; he gave her mustard and water, and made her sick. At the hospital, one hour after taking the poison, the face was flushed; breathing natural; pupils moderately contracted; she seemed to have little the matter with her; but while there she became a little drowsy, and the pupils were more contracted. The stomach was washed out with water, and she became brighter and less drowsy. July 3rd: Well; discharged in the afternoon.

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CASE 29.—1904, M. æt. 73.—August 6th: Said to have taken five ounces of laudanum. In surgery, rather drowsy; but conscious; pupils very small, equal, contracting to light. Washed out; the washings dark green, smelling of opium. Two ounces of castor oil were given through the tube. In the ward, pulse, 80 to 100; respirations, 20. Five minims of liquor atropinæ were injected subcutaneously. August 8th: Condition satisfactory; pupils widely dilated. August 9th: Scarlatiniform and erythematous rash. Last night was very lively, trying to get out of bed, using unparliamentary language. August 11th: Rash fading. August 12th: Went out.

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CASE 30.—1904, F. æt. 30.—Is in the habit of taking opium. She took on December 20th two ounces of some mixture in which were two drachms of nepoche. Two hours after admission she was extremely drowsy, but the pupils were not much contracted. In the ward vomited after a mustard emetic, and then had a pint of strong coffee. Complained of pain in lower part of abdomen. The pupils were more contracted four hours later. December 21st: Went out.

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CASE 31.—1905, F. æt. 53.—February 14th: Took an ounce of chlorodyne (equal to 4 grains of morphia) and 2 ounces of spirit of salts. C.O.A.: Appeared to be asleep, but could be made to speak; vomited greenish-brown fluid shortly after admission; pupils contracted; epigastric pain. Some solution of sodium bicarbonate was given, and she vomited. She was then washed out with about 80 ounces of solution of potassium permanganate. Three minims of atropine solution were

injected hypodermically, and repeated after half an hour. February 15th: Quite conscious; dry throat. Some pain and swelling at the site of the injections; otherwise well; went out February 21st.

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CASE 32.—1905, M. æt. 55.—July 4th: Bought three pennyworth of laudanum, rubbed his joints with some and swallowed the rest. Brought up by the police at 4 p.m.; then drowsy; can be roused to speak, but soon falls asleep again; pin-point pupils; pulse slow; knee-jerks absent. Washed out with solution of potassium permanganate of .1 per cent. strength. Atropine solution, 3 minims, injected subcutaneously. He became more drowsy, and the lavage and atropine were repeated. Until 5 p.m. constant attention was necessary to keep him awake. Then the pupils dilated; he became suspicious and excited. At 9 p.m. he tried to get out of bed, shouted and struggled; then he slept, but was noisy when waked. Was transferred to the strong room. He was quieter at night, but dusky; sweating. Examination showed a large liver, and râles at the bases of both lungs. He was restless and "saw persons" for two days more. On July 8th: Was well, and went out July 10th.

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CASE 33.—1905, M. æt. 65.—Suffers from the pains and gastric crises of tabes dorsalis, and has acquired the morphia habit. On August 4th had severe pains, took a draught, and was found by the police in the street. C.O.A.: Semi-comatose, with slow respiration; wants to be let alone; pin-point pupils. Was washed out with solution of potassium permanganate, and had two injections of atropine, 1/100 grain, with an hour's interval. In three hours he was better, less drowsy; pupils dilating. August 5th: Quite well.

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CASE 34.—1905, M. æt. 61.—On December 2nd took an ounce of laudanum and two drachms of oxalic acid. Found sitting in a chair over the fire, and confessed what he had done; then vomited. C.O.A.: 7 p.m.: Coma; breathing heavily; slight cyanosis, pupils very contracted; pulse, 96; respirations, 16. Stomach washed out twice; the first washings smelt of opium. Twenty grains of caffeine injected subcutaneously; was then walked about from 9.30 to 11.30, with short intervals, during which he was able to converse, though he did not know where he was. At 12 p.m. was less drowsy. At 12.30 was allowed to go to bed. December 3rd: Much better; pulse, 100; respirations, 20. December 4th: Went out.

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CASE 35.—1906, M. æt. 32.—Took two drachms of laudanum; two hours later in the surgery had half a pint of salt and water, and was sick. In the ward was slightly drowsy, skin cold and moist; pulse small; no marked signs of opium poisoning. Hot coffee was given, and 3 minims of

liquor atropinæ were injected. He was roused constantly, and ammonia was applied to the nostrils. At 12 p.m. he was more drowsy; pulse feeble. 1 a.m.: Another injection of 3 minims of liquor atropinæ and 2 minims of liquor strychninæ. Slept from 4 a.m. till noon. Well.

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CASE 36.—1906, M. æt. 40.—Was found asleep outside the hospital, and said that he had taken by mistake one drachm of powdered opium. He was a West Indian, and used to take opium and quinine for malaria. C.O.A.: Four hours after taking the opium, sleepy but easily roused, and was able to walk to the ward without assistance. He had vomited in the surgery after an emetic. Admitted at 4 a.m., he slept soundly, and was quite well later in the same day.

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CASE 37.—1906, M. æt. 47.—The patient was found in his room in a comatose condition. A medical man was sent for; he found the mark of a recent injection in the skin of his arm, but no syringe or bottle could be found. The doctor washed him out and sent him to the hospital. On arrival he was comatose, with cyanosed face, stertorous, slow, and irregular breathing, absent reflexes, pin-point pupils, and subnormal temperature. Three minims of liquor atropinæ were injected subcutaneously, and he was taken to the wards, where he was stimulated by smacking and similar means. It was observed that he bruised very readily; otherwise he did not respond at all. The galvanic battery was applied for about 20 minutes, and the breathing became more regular, and the corneal reflex returned. Oxygen inhalations and a hypodermic injection of 10 minims of liquor strychninæ were given; and subsequently a hot coffee enema, but his breathing became bad again, and the reflexes failed. He was again faradised, and the breathing, the pulse, and the reflexes improved. Ether, 30 minims, subcutaneously, and oxygen inhalations were also given. He went on well for some time, till 4 a.m., when his pulse began to fail, and he died ten minutes later. Post-mortem, morphia was found in the stomach, and was also present in the fluids washed out from the stomach.

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CASE 38.—1906, M. æt. 67.—Was admitted on December 7th, 1906, said to have swallowed four pennyworth of laudanum. His friends gave him salt and water, and he was sick frequently after it. C.O.A.: Very drowsy, but quite sensible; can walk and talk; pupils small, but react to light. He was washed out with solution of potassium permanganate, and 3 minims of liquor atropinæ were injected, and a coffee enema. He went out next day quite well.

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CASE 39.—1907, F. æt. 16.—Took half a pint of lead and opium lotion. At 7.30 p.m. she was in the surgery, wide awake and tearful; hands cold; pulse small; pupils rather small, reacting to light. She

was washed out with solution of potassium permanganate, 1 drachm to a gallon (.08 per cent.). This was repeated in half an hour, and half a pint of the solution was left in the stomach. During the process, however, she vomited two or three times. She was put to bed; the pupils became smaller; and she became drowsy. An enema of coffee and port wine was given, and a mixture of ammonia and ether by the mouth. A nurse was in attendance to prevent her sleeping, but she became more drowsy, and smelling salts were used constantly. Three minims of liquor atropinæ were injected, and she was less drowsy with wider pupils half an hour later. White mixture was then given her. At 12 p.m. she was again drowsy, and she was kept awake with great difficulty. A few hours later it was necessary to keep her walking to prevent her from sleeping, and this was done for an hour or more. In the later morning she was allowed to sleep naturally, and two days later went out well.

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CASE 40.—1907, M. æt. 52.—At 6 p.m. the patient took an ounce of tincture of opium. At 11 p.m. he felt sick and tired, and went to the police, who brought him to the hospital at 11.45. He had then pin-point pupils, but otherwise seemed little affected. He was not at all inclined to be drowsy; his pulse was good, and there was no collapse. He was washed out with solution of potassium permanganate of .16 per cent. strength; later some castor oil was given. There was no drowsiness all night, and he went out well next day.

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CASE 41.—1907, M. æt. 35.—Took three pennyworth of tincture of opium (? 2 drachms), and appears to have gone at once to a public-house and told them to send for the police. C.O.A.: Drowsy; muscles relaxed; pulse and respiration normal; pin-point pupils. Three minims of liquor atropinæ injected. "Washed out generously with potassium permanganate." Two hours later was again washed out with the same. He kept awake all the rest of the night without any difficulty.

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CASE 42.—1907, M. æt. 61.—Took two pennyworth of tincture of opium, and placed himself on a railway line in order to be run over, but he was seen in time, the train was slowed, and only pushed him off the line. On arrival: Drowsy, but sensible; pulse slow and full; pupils extremely contracted. In the surgery his stomach was washed out with solution of potassium permanganate, and 10 ounces were left in. In the ward 3 minims of liquor atropinæ were injected. Next day he was well, and two days later went out.

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CASE 43.—1907, M. æt. 23.—Took an ounce of laudanum. He was very cold and collapsed, and the pupils were small. He was washed out in the surgery, and soon improved.



CASE 44.—1907, M. æt. 35.—Brought to the surgery by the police ; supposed to have taken half an ounce of tincture of opium. The pupils were small, otherwise he was quite well.

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CASE 45.—1907, F. æt. 36.—Having quarrelled with her husband, she took two pennyworth of laudanum. She was found by the police. The stomach was washed out at the police station, and she was brought to the hospital. On arrival, she appeared to be in a condition rather of drunkenness than of opium poisoning. She said she was very sleepy, but this was hardly evident of itself. Three pints of a solution of sodium bicarbonate were given to her, and she vomited freely. She slept badly, complained of headache, and soon recovered.

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CASE 46.—1907, M. æt. 1½.—Had taken 3 drachms of syrup of poppies; it was never drowsy or sick. On admission its pulse was 144. The child was often standing up in its cot, crying vigorously, with widely dilated pupils.

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CASE 47.—1908, F. æt. 17.—Swallowed about half a bottle of lead and opium lotion supplied from the hospital. She came back at once, and a mixture of sodium sulphate, 1 ounce, sodium bicarbonate, 1 ounce, and sodium chloride, 1 drachm, in a pint of water, was given to her. She was sleepy ; did not vomit. On the stomach tube being passed she was sick; but she was washed out with solution of potassium permanganate. Coming into the ward she was drowsy, but the pupils were normal. Half a pint of coffee was given by the mouth. Later she was still drowsy. She was again washed out, and 2 ounces of white mixture (*mistura magnesiae cum magnesii sulphate*) were put into the stomach. She had more hot coffee during the night. She slept a good deal and recovered.

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CASE 48.—1908, M. æt. 28.—Was said to have taken 2 ounces of tincture of opium ; was found by the police asleep on the pavement. On admission was sleepy ; pupils were not contracted ; knee-jerks were present. He was washed out with solution of potassium permanganate of .16 per cent. strength, and had in succession half a pint of hot strong coffee, an inhalation of amyl nitrite, which improved the pulse, injection of 3 minims of liquor atropinae, injection of ether, 10 or 20 minims, another injection of atropine, an enema of port wine and coffee, and *mistura ammoniae et ætheris*. He was better the next day, and went out the day following that.

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CASE 49.—1909, F. æt. 49.—Had taken two pennyworth of tincture of opium. The police gave her a salt and water emetic, and on admission she had no sign of opium poisoning other than contracted pupils.

CASE 50.—1910, M. æt. 56.—He was said to have taken 3 drachms of tincture of opium. He came to the surgery, and was watched for two or three hours, and then allowed to go out. He appears to have become drowsy afterwards, and the police brought him back. On arrival he was sleepy and difficult to rouse. He was washed out with mustard and water, and then with a solution of potassium permanganate, of which 6 ounces were left in the stomach. An injection of 2 minims of liquor atropinæ sulphatis was given under the skin, and of strong coffee per rectum; later several injections subcutaneously. He soon recovered from all signs of opium poisoning.

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CASE 51.—1910, M. æt. 33.—Took some horse colic medicine. Two hours later he was drowsy; vomited in the surgery; the pupils were pin-point in size. He was washed out; and some colocynth and hyoscyamus pill was given to him. The next day he had some headache and was drowsy: the pupils were larger; and he recovered.

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CASE 52.—1910, F. aged seven months.—Had had soothing powder. Twenty minutes later vomited and became drowsy. On admission was comatose, the pupils contracted, the pulse very small indeed. She was washed out with solution of potassium permanganate, and 1 ounce was left in the stomach. Four minims of ether were injected subcutaneously. She quickly improved after this injection.

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CASE 53.—1910, M. æt. 25.—He took 2 ounces of laudanum at 6.10 p.m., and was brought to Guy's Hospital at 6.30. He was very sleepy; he was rubbed and slapped; and the stomach was washed out with one gallon of dilute solution of potassium permanganate, of which 6 ounces were allowed to remain; and half a pint of coffee was introduced as well. Three minims of liquor atropinæ were also injected subcutaneously. Admitted into the ward he was less sleepy; the pupils not very small; pulse rapid and irregular. A similar dose of atropine was administered, and one pint of black coffee was ordered hourly per rectum, but there is no note as to how many pints were employed. He "was with difficulty kept awake, and later on had to be walked about." He recovered.

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CASE 54.—1910, F. æt. 45.—She had taken a quantity of beer and some laudanum, but the amount of either does not seem to have been known. On admission she was perfectly conscious, but talked like a drunken woman. The pupils were contracted and did not react to light or to accommodation. She remained for a time half drunk without becoming more drowsy, and recovered without any antidote for opium being employed.

CASE 55.—1911, M. et. 44.—On September 20th, at 10 p.m., he took four pennyworth of laudanum, equal to 6 drachms. A quarter of an hour afterwards he was found by the police cold and comatose. They aroused him with some difficulty, and induced him to take some salt and water, which he vomited. At 10.30 he was in the surgery, and three pints of a solution of potassium permanganate of .2 per cent. strength were passed through his stomach; and an injection of 3 minims of liquor atropinæ was given. He was then walked to the ward. There he was again washed out, put to bed with blankets and hot bottles, and a pint of hot coffee was given per rectum. The pupils were not very contracted; he talked cheerily and incessantly, but was seized by an overpowering wish to sleep at times. This was prevented throughout the night. No more alarming symptoms occurred, but he complained of numbness and coldness of the limbs, and of transient abdominal pain. On September 21st: Quite sensible, but he has some headache and occasional "fits of shaking." He had a high colon wash out with saline solution, and the shaking stopped. He was allowed to sleep from 11 p.m. to 5 a.m., and on September 23rd he still had headache, and was very weak. He had a few more shiverings. September 25th: Occasional cramps in the legs, and feeling of weakness in the limbs. Headache absent. Anæsthesia of the surface from Poupart's ligament to the level of the umbilicus on each side. Daily enema to open bowels. September 29th: Still constipated; going out.

# NOTES FROM THE THROAT DEPARTMENT.

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By

F. J. STEWARD AND W. M. MOLLISON.

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## 1. CASES OF MALIGNANT DISEASE OF ŒSOPHAGUS AND PHARYNX.

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DURING the year 1909 twelve cases of œsophageal obstruction attended in the Out-Patient Department; of these only one could be traced to his death, and one case, a girl of 22, was probably not a genuine case of obstruction at all. All attempts at tracing the patients by writing to the various doctors who sent the cases to hospital failed except in one case, no doubt on account of the length of time that had elapsed since the patients' attendance in the Out-Patient Department, and on account of the habits patients have of changing their places of residence, and lastly, because most, if not all, had died.

The cases are tabulated with those of 1910. It will be noticed that the various points to which attention is drawn in connection with the 1910 cases hold for the 1909 cases, with one exception. In 1910 only one case showed paralysis of the larynx, and in that case the left cord was paralysed.

During the year 1910 thirteen cases of epithelioma of the œsophagus attended the Throat Out-Patient Department. Of these, 10 have been traced, and all died; one of these is known to have gone to an infirmary, and there is no doubt that death took place soon after. The remaining 3 cannot be traced, and will not be considered further.

A complete analysis of the cases is appended, and the cases will be referred by the number attached to each. Of the 10 cases 9 were men, the one woman was aged 35, and the growth was one of those occurring on the party wall between the larynx and hypopharynx. The average age of the men was 61·8 years; except for one of 82, all were between 53 and 67.

The disease is a rapid one. The time that elapsed between the onset of symptoms and death varied from 2 months to 27 months; the average gives 9·5 months, or, excluding the case of 27 months, the average was only 6·8 months.

The *cause* of death. Four cases (1, 4, 8, 10) were operated upon; three had gastrostomy performed and one a jejunostomy, because the stomach was too small for gastrostomy, and the growth involved the cardiac end of the stomach. In Case 1 the patient improved after operation and left the hospital; he attended in the Out-Patient Department for a time, and eventually went to an infirmary, and doubtless died shortly after. In Case 4, after jejunostomy, patient survived four months, but died in hospital. Case 8, the patient died shortly after gastrostomy from septic broncho-pneumonia. At the post-mortem examination the growth was found at the level of the cricoid and had ulcerated into the trachea. Case 10, after gastrostomy, died with signs of general peritonitis. At the post-mortem examination growth was found at the junction of pharynx and œsophagus, but was not ulcerated.

Six cases (2, 3, 5, 6, 9, 11) were not operated upon; of these, four (2, 3, 6, 11) died at home; two cases (5 and 9) died in the hospital; Case 5 had as his chief symptom violent coughing whenever he made an effort to swallow. At the post-mortem examination broncho-pneumonia was found, and there was a mass of growth at the junction of the pharynx and œsophagus, but no mention is made of ulceration; there were secondary deposits in the liver. Case 9 died in hospital, his chief symptom latterly being violent cough on attempting to swallow: the patient died suddenly. Post-mortem examination showed growth at the level of the bifurcation of the trachea and perforation

through to the trachea. The four cases that died at home were traced through their doctors.

Dr. Carter kindly wrote of Case 2 that he died of exhaustion one month after being seen in the Out-Patient Department.

Dr. Beven kindly wrote about Case 6: "The patient died three months after he was seen in the Out-Patient Department from complete obstruction and exhaustion."

Dr. A. G. Butler, of Penge, most kindly made inquiries, and discovered that Case 11 died thirteen months after he attended in the Out-Patient Department, but the cause of death could not be ascertained.

Dr. Bryden gave a very full account of Case 3 that was under his care till death. The man died of starvation. For some time he was fed through a rubber tube passed through the stricture, but latterly the passage of the tube was very difficult, and feeding led to choking.

Reviewing the cases from the point of view of the cause of death, it is noticed that broncho-pneumonia, due to ulceration of the growth into the trachea, is the most frequent cause, and that starvation and exhaustion play a prominent part. Five cases were examined post-mortem; in only one were any secondary deposits found, and when found (Case 5) they were in the liver.

The position of the stricture was located in seven cases; of these, five were seen post-mortem. In four cases the stricture was found in the upper part of the oesophagus about 10 inches from the teeth. In one case it was at the level of the bifurcation of the trachea, in one case at the lower end of the oesophagus, and in one on the party wall, i.e., the posterior aspect of the cricoid cartilage.

*The Symptoms.*—All the male patients complained of difficulty in swallowing for a longer or shorter time, and all of them stated they could not swallow solids. In the case of one man (10) this statement was modified to inability to swallow solids unless washed down with liquid, and in another (1) to scarcely swallow solids. Four men (1, 2, 4, 9) volunteered the fact that

they had lost weight; one man (3) definitely said he had not lost weight; another (5) was not sure about it; in the remaining four no loss was mentioned, though doubtless it was quite definite as judged from the subsequent history.

Two patients (5 and 8) complained of vomiting; no doubt in Case 8 the accompanying cough, due to ulceration of the growth, was the main symptom, and induced the retching. Pain in the chest was complained of in two cases (3 and 10), and in one case (2) the patient pointed to the cricoid as the seat of obstruction. Change in the voice was noticed in the three men in whom one cord was paralysed.

The four cases in which paralysis of cord occurred deserve more notice. In Case 2 the right cord was paralysed; in Case 4 the left cord was paralysed; here the growth was at the lower end of the œsophagus, and the paralysis is difficult to explain, particularly as a post-mortem examination revealed no gland pressing on the nerve nor any aneurysm. In Case 7 the right cord was paralysed by direct involvement in the mass of growth. In Case 10 the left cord was paralysed. The diagnosis of this case is not quite certain, though probable; the mass seen behind the sternum by the X-ray examination was apparently not an aneurysm, but a mass of growth. Case 7, that of the woman with the "party wall" growth, had as the chief symptom a mass in the neck which was tender and had grown rapidly. The striking feature about this case was the very large mass in the neck compared with the original growth, which, in its turn, had ulcerated into the trachea.

X-ray examination was carried out in three cases (6, 8, 10). The patient was given a mouthful of bismuth made into a thin paste with water, and examination made with the screen of the œsophagus as the mouthful was swallowed. In Case 6 no obstruction was seen at all, though the patient had for two months only been able to swallow liquids, and three months later died from complete obstruction. In Case 8 there was no obstruction seen beyond what is normal opposite the arch of the aorta. This patient had been unable to swallow for twelve days, and two months later died, a growth being found at the level

of the bifurcation of the trachea, ulcerating into the trachea. Judged by these two cases, and this particular method of administration of bismuth, X-ray examination of oesophageal obstruction is no help towards a diagnosis. In Case 10 the X-ray examination revealed a mass behind the sternum which was apparently not an aneurysm, but a mass of growth. Apart from these particular cases it may be mentioned that X-ray examination has now for some years been made in all cases of oesophageal obstruction coming to the Throat Department as a matter of routine. The results obtained have made it clear that this method of examination is seldom of assistance either in detecting the presence of a stricture or in locating it, except in cases where the stenosis is already extreme. On the other hand, the findings have, on occasion, been decidedly misleading.

In two cases (3 and 5) direct examination of the oesophagus by Killian's tubes was most useful as an aid to treatment. In both cases bougies could be passed through the stricture under the eye of the operator where previously no bougie could be passed. In Case 3, subsequent to the passage of the bougie, the patient was able to have a gastric tube passed for feeding, and was made more comfortable for three months. In Case 5 the passage of bougies was so effective that the patient could even for a time swallow solids.

*Operation.*—Four cases were submitted to operation: three had gastrostomy performed, one jejunostomy on account of the small size of the stomach and the involvement of the cardiac end of the stomach in the growth. Three died in hospital shortly after operation; the fourth case went out of hospital and attended for a few weeks in the Out-Patient Department, and then went to an infirmary as he was obviously going down hill.

These results are not good, but it must be remembered that the patients only attend when their condition is already very grave and that they cannot get suitable liquid food at home, so that the operation is undertaken as a last resort upon a patient already suffering from prolonged starvation; and, in consequence, these patients have no resistance, and are quite unable to withstand the shock of an abdominal operation.



No.	Name and Age.	Date of First Attendance.	Symptoms.	Position of Growth.	Duration.	Post-Mortem Examination.	Treatment.	Result.	Paralysis of Cords.
1	John L., 61	Jan., 1909	Difficulty in swallowing. Can only take fluids. Wasting	Lower end of œsophagus	2 months	—	—	—	Nil
2	Alfred B., 37	April, 1909	Difficulty in swallowing. Can take soft solids	?	9 months	—	—	—	Nil
3	Wm. J., 55	July, 1909	Can swallow fluids only. Has lost 2 stones in weight	?	2 months	—	—	—	Nil
4	Walter L., 57	Nov., 1909	Difficulty in swallowing. Loss of weight. Aphonia	? 16in. from teeth	2 months	—	—	—	Left cord paralysed
5	Chas. H., 53	July, 1909	Difficulty in swallowing solids. No loss of weight	?	1 month	—	—	—	Nil
6	Joseph T., 64	May, 1909	Cannot swallow solids. Pain between shoulders. Wasting	?	4 months	—	—	—	Nil

No.	Name and Age.	Date of First Attendance.	Symptoms.	Position of Growth.	Duration.	Post-Mortem Examination.	Treatment.	Result.	Paralysis of Cords.
7	Prudence V., 22	Oct., 1909	Difficulty in swallowing. No loss of weight	None found. Full-sized bougie passed	8 months	—	Bougie	—	Nil
8	Robert F., 70	Aug., 1909	Sore throat. Slight difficulty in swallowing. Pain right ear	Right pyriform sinus. Glands	6 months	—	—	—	Nil
9	Henry B., 65	May, 1909	Irritation of throat. No pain. Feeling of a lump in throat	Lower end of pharynx	Some months	—	—	—	Nil
10	Anne S., 35	Oct., 1909	Can only swallow fluids and soft solids. Loss of weight	Lower part of pharynx	6 months	—	—	—	Nil
11	John W., 52	Oct., 1909	Can only swallow fluids. Cough. Alteration of voice	Left pyriform sinus. Glands	2 months	—	Tracheotomy	Died Nov. 30th, 1909	Nil
12	Chas. N., 42	Sept., 1909	Pain and difficulty in swallowing. Voice altered. Coughs up blood. Loss of weight	Lower end of pharynx	6 weeks	—	—	—	Nil

No.	Name and Age.	Date of First Attendance.	Symptoms.	Position of Growth.	Duration.	Post-Mortem Examination.	Treatment.	Result.	Paralysis of Cords.
1	Alfred W., 53	May, 1910	Difficulty in swallowing for 6 months. Can scarcely swallow solids. Has lost much weight	12 in. from teeth	Probably about 10 months	?	Gastrostomy in July, 1910	Died in infirmary (?)	Nil
2	James H., 82	June, 1910	Difficulty in swallowing 6 months. No solids for 2 months. Loss of weight. Points to cricoid as seat of obstruction. Voice now deep, now squeaky. Right vocal cord paralysed	Probably just below cricoid	7 months	?	Nil	Died July, 1910 (Dr. Carter)	Right cord paralysed
3	Chas. D., 56	April, 1910	Difficulty in swallowing 4 months. Only taken fluids for past month. Not lost weight. Pain behind sternum. Larynx normal	9 in. from teeth	7 months	No growth felt anywhere, but no P.M.	Bougies passed through Killian's tubes. Subsequently fed through tube. Choking prominent symptom	Died at home Aug. 20th (Dr. Bryden)	Nil
4	Fred. H., 56	May, 1910	Difficulty in swallowing for 10 weeks. Can only swallow fluids and custard. Difficulty in swallowing saliva. Has lost weight. Voice changed 2 weeks ago. Left cord paralysed. Why?	Lower end of oesophagus and cardiac end of stomach	8 months	Growth at lower end of oss	Jejunostomy, as stomach very small and involved in growth	Died subsequent to operation	Left cord paralysed

No.	Name and Age.	Date of First Attendance.	Symptoms.	Position of Growth.	Duration.	Post-Mortem Examination.	Treatment.	Result.	Paralysis of Cord.
5	Robert Y., 67	July, 1910	Difficulty in swallowing 5 weeks. No solids can be taken, as they are at once vomited. Not lost weight (?). Larynx normal	25 c.m. from teeth. 10 in.	10 months	Growth at junction of pharynx and cesophagus. Broncho-pneumonia. Secondary deposit in liver	Examination with Killian's tubes. Piece of growth removed for microscopic examination. Bougies passed. Much improvement. Subsequently could swallow solids	Died in hospital	Nil
6	Henry G., 62	July, 1910	Difficulty in swallowing off and on for 2 years. Worse during last 2 months, and now able to swallow liquids only. X-ray showed no obstruction at all. No delay in passage of bismuth	—	27 mths.	?	—	Died Oct., 1910, with complete obstruction and exhaustion (Dr. Beven)	Nil
7	Nellie S., 35	Sept., 1910	Mass in right side of neck, tender, and has grown rapidly last 2 months. ? lost weight	Party wall	? 5 mths. ? history	Growth of cesophagus at level of cricoid. Indirect connection with mass in neck. Ulceration into trachea. Broncho-pneumonia	Gastrostomy	Died after operation	Right cord paralysed

No.	Name and Age.	Date of First Attendance.	Symptoms.	Position of Growth.	Duration.	Post-Mortem Examination.	Treatment.	Result.	Paralysis of Cords.
8	Fred. H., 56	Nov., 1910	Inability to swallow for 12 days. Nausea, retching. Coughing on trying to swallow. X-ray—no obstruction except opposite aortic arch (normal)	Level of bifurcation of trachea	? 4 mths.	Growth at level of bifurcation of trachea. Ulceration to trachea. Broncho-pneumonia	Too ill for any operation	Died suddenly in hospital, Jan. 27th, 1911	Nil
9	Alf. W., 60	Sept., 1910	Inability to swallow solid food. Has lost weight	Junction of ph. and oss, 8-9 in.	? 2 mths.	Growth at junction of pharynx and oesophagus. No ulcer. Early peritonitis	Gastrostomy	Died in hospital after operation	Nil (?) Larynx could not be seen
10	Fred. B., 65	Oct., 1910	Difficulty in swallowing. Can only swallow solids with liquids to wash them down. Hoarseness. Pain across chest. X-ray—swelling behind upper part of sternum. No pulsation	?	? 15 mths.	? ?	?	Died Nov., 1911 ? cause (Dr. A. G. Butler)	Left cord paralysed

## 2. ANALYSIS OF THE CASES WHICH ATTENDED THE THROAT DEPARTMENT IN 1909 AND 1910.

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The object of this analysis is to show the relative frequency of the various affections of the throat and nose.

The total number of cases was 5,424; of this total more than one-half were patients (mostly children) suffering from symptoms due to the presence of enlarged tonsils and adenoids.

### *Nose—*

Foreign body in, 4.  
Epistaxis, 29  
Anosmia without any obvious cause, 4.  
Hay fever; Sneezing, 6.  
Rhinorrhœa, 8.  
Depressed bridge, 5.  
Swollen, no cause found, 4.  
Abscess on, 2.  
Habit obstruction, 2.  
Lupus, 8.

### *Septum—*

Deflected and producing symptoms, 156.  
Hæmatoma of, 2.  
Perforated, 4.  
Abscess, 1.  
Sarcoma, 1.

### *Rhinitis—*

Acute, 14.  
Chronic simple, 157.  
    Vaso-motor, 7.  
    Due to snuff, 1.  
Atrophic, 116  
Membranous, 1.

Diphtheritic, 14.

Tuberculous, 8.

Syphilitic (tertiary), 12.

*Hypertrophied Turbinals—*

Inferior, anterior ends, 176.

Posterior ends, 8.

Middle, anterior ends, 12.

*\*Malignant Polypus, 1.*

*Sinus Disease—*

Antrum, 24.

Malignant tumour, 1.

Frontal, 16.

Ethmoid, 10.

Not limited to one sinus, 76.

*Nasopharynx—*

Adenoids, with or without enlarged tonsils, 3,014.

Post nasal catarrh, 41.

*Mouth—*

†Swollen upper lip, 1.

Carious teeth, 49.

Salivary calculus, 2.

Long uvula causing symptoms, 7.

Polypus of uvula, 1.

Paralysis of soft palate (post diphtheritic), 6.

Stomatitis, simple, 4.

Secondary, syphilitic, 5.

Herpes, 1.

\* This case was that of a boy, Sidney G., aged 14. He was admitted under the care of Mr. Dunn. Superior maxilla excised; re-operation for hæmorrhage; applications of radium; patient is alive and well.

† A girl of 7; much swelling, probably septic in origin; resisted all efforts at treatment.

*Pharynx—*

- Foreign body in, 3.
- Globus hystericus, 32.
- Pharyngitis, simple, 91.
  - Sicca, 3.
  - Secondary syphilitic, 13.
  - Tertiary syphilitic, 32.
- Diphtheria, 6.
- Tonsilitis, acute and chronic, 169.
- Quinsy, 29.
- Epithelioma of anterior faucial pillar, 1.
- Papilloma, 1.

*Larynx—*

- Foreign body in (tintack, patient refused admission), 1.
- Inspiratory stridor, cause not to be found, 2.
- Laryngitis, simple, 60.
  - Tuberculous, 26.
  - Sicca, 4.
  - Pachydermia, 15.
  - Tertiary syphilitic, 5.
- Papilloma of vocal cord, 3.
- Fibroma of, 1.
- Paralysis of cords, functional, 19.
  - Abductor, 5.
  - Fixation of arytaenoid, 2.
  - Edema of arytaenoid, 1.
  - Malignant disease involvement, direct, 7.
  - Indirect, 6.

*Œsophagus—*

- Obstruction by malignant disease.
  - These cases are dealt with in a separate analysis.



## 3. CASES OF LARYNGEAL PARALYSIS SEEN IN 1910.

DURING the year 1910 there were ten cases of fixation or paralysis of one or both vocal cords seen in the Throat Department, and among these examples of many of the known causes. These ten cases were made up as follows:—

Three cases of nerve involvement by epithelioma of œsophagus. In two of these cases the right cord was paralysed, in the third the left cord.

One case of direct involvement of the left cord by a pharyngeal growth; probably there were other similar cases, but the notes do not mention the fact in other cases of pharyngeal growth.

One case of involvement of the left cord by a mediastinal tumour or perhaps an aneurysm.

One case of fixation of the left cord by involvement of the arytaenoid cartilage, probably the result of previous tuberculous ulceration. The patient had signs of old phthisis at the apex of the left lung, and there was the suggestion of involvement of the left recurrent nerve in the thickened pleura.

One case of partial fixation of the left cord by inflammatory swelling of the arytaenoid cartilage. The cartilage was œdematous and paralysed.

One case of fixation of the left cord by old tertiary syphilitic disease of the arytaenoid.

Two cases of bilateral abductor paralysis, both due to tabes; in one case the disease had been diagnosed in the Neurological Department, and the man was sent to the Laryngological Department for an opinion on the laryngeal condition. The other case was of greater interest in that the paralysis of the cords was the prominent sign of his disease, the only other sign confirmatory of tabes being absence of both tendo-Achillis-jerks; knee-jerks were present, and the pupils reacted to light and accommodation. Doubtless this case would have been demonstrated as one showing the cord paralysis to be the earliest sign before attention had been drawn so prominently to the value of the lost tendo-Achillis-jerk.

Among these cases of laryngeal paralysis may be included nine of functional paralysis.

# REPORTS FROM THE AURAL DEPARTMENT.

## ON THE RESULTS OF HEARING TESTS ON TWENTY-FIVE NORMAL INDIVIDUALS.

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By

W. M. MOLLISON.

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THE following investigation was undertaken to ascertain whether a standard could be obtained for the various recognised tests used for the determination of different forms of deafness.

Most clinics use a certain number of well-known tests—Weber's, Schwabach's, and Rinné's; but it is now recognised that these tests alone give but a rough idea of the form of deafness from which a patient is suffering, and in order to get more comprehensive results and more assistance towards a diagnosis the extremities of the audible scale should be investigated.

The normal ear can appreciate sounds of a very widely different wave length from a note given by 16 vibrations per second to one of, roughly, 26,000 vibrations per second. The whole series of perceptible notes is spoken of as the "auditory field," a term analogous to the "visual field"; the lowest and highest notes heard are spoken of as the "lower" and "upper tone limits."

To produce the notes of the auditory field, a series of tuning forks is used, as suggested by Bezold, "Bezold's continuous series of tuning forks." They range from a very large fork,  $G_2$ , of 16 vibrations per second to  $c^5$  of 4,096 vibrations per second, and between these there are  $C_1$ ,  $G_1$ ,  $C$ ,  $G$ ,  $c$ ,  $g$ ,  $c^1$ ,  $g^1$ ,  $c^2$ ,  $g^2$ ,  $c^3$ ,  $g^3$ ,  $c^4$ ,  $g^4$ .

This series of forks, made by Prof. Edelmann of Munich, is recognised as the standard series, and is in use at all recognised clinics. Above  $c^5$  tuning forks are not reliable, and to produce notes from this pitch to the highest audible, various apparatus have been suggested. That most commonly used is *Galton's whistle*.

This apparatus is a small closed organ pipe, wind being blown in by a small rubber ball. The whistle has been modified by Edelmann, and the notes produced are accurately estimated by means of a "sensitive" flame. A table is supplied with the instrument, so that by adjusting the length of the pipe accurately to an affixed scale, notes of known vibration frequency are produced.

Another method of producing the highest notes is a *monochord*. This consists of a wire stretched horizontally on a frame, and made to vibrate in its length by rubbing with a piece of wash-leather covered with resin. The effective length of the wire can be altered by a screw sliding on the frame, and the note produced is stated on a scale beneath this screw. In an improved form the wire is stretched on a metal frame; that in use at Guy's is on a wooden frame (Schultze's) and is, perhaps, more difficult to use.

The monochord is said by some observers to give far more accurate and constant results than Galton's whistle, but there are difficulties in its use. First, the very great mechanical difficulty in the production of the note, and second, the marked "nebenegeräusch" when the wire is rubbed, making the appreciation of the note at all very difficult, and third, its use requires much practice. This last is an objection which cannot be urged against Galton's whistle.

The upper tone limit was investigated by means of the monochord in a certain number of cases, but the difficulties in its use and the difficulty experienced by the observed persons who were all scientifically trained in detecting the highest notes, led to its use being abandoned.

It will be noticed that in all the cases where it was used the note heard was lower than that heard with Galton's whistle.

but it should be noticed also that in no case was there any marked disparity between the results given by the two instruments, and in all cases the differences were much the same. As far as the small number of observations is concerned, Galton's whistle is as reliable for the fixation of the upper tone limit as the monochord, and quite simple to use.

Lower tone limit.	AUDITORY FIELD.																Upper tone limit.
	G <sub>2</sub>	C <sub>1</sub>	G <sub>1</sub>	C	G	c	g	c <sup>1</sup>	g <sup>1</sup>	c <sup>2</sup>	g <sup>2</sup>	c <sup>3</sup>	g <sup>3</sup>	c <sup>4</sup>	g <sup>4</sup>	c <sup>5</sup>	
(16 vibrations per second.)	(Bezold's Tuning Fork Series.)																28000
																	(Galton's Whistle or Monochord.)

Besides noting the lower and upper limits of the auditory field, Schwabach's and Rinne's tests were carefully carried out by the following methods:—

*Schwabach's Test.*—This is a test for the investigation of the conduction of sound through the bone. The test is designed to discover whether the conduction through the bone is better or worse than in a normal individual; to compare a patient's perception with the observer's is a little awkward, and presupposes a normal bone conduction in the observer. For this test a fork of 108.75 vibrations was used with a special attachment by means of which it is possible to note when the vibrations reach a certain intensity. This attachment makes it possible to note the amount of bone conduction in terms of time, and thus does away with the difficulty. At the moment the fork has reached the intensity represented by the special attachment, it is allowed to rest on the patient's head by its own weight, and at the same moment a stop-watch is started: the moment the patient ceases to hear the fork the watch is stopped and the time noted. It is obvious that the time during which the fork is heard will vary according to the

intensity at the beginning of the observation, and as each "attachment" will vary a little, each fork must be standardised by observations on normal individuals before it can be used in abnormal cases.

*Weber's Test.*—A sounding fork is placed on the head in the mid line; normally the fork is appreciated as much in one ear as in the other. Should any fault in the conducting apparatus of one ear be present, the fork is heard more plainly in that ear than the other.

*Rinné's Test.*—This test is designed to compare the capacity to hear through the bone of the mastoid process with that through the air in the meatus. For this test a small "a" fork is used; it has the advantage that the sound made by it carries but a short distance through the air. To carry out the test the foot of the fork is rested on the mastoid process, and when the sound is no longer heard, the extremity of the fork is held in front of the external auditory meatus, and, in normal persons, the note is again heard; this result is spoken of as "positive."

In this investigation the actual time during which the fork was heard through the air after it had ceased being heard through the bone was noted: thus, the result is expressed—Rinné, right, +40 sec.; left, +42 sec.

It has been argued that this test is of little value, as it gives the result that air conduction is better than bone, whereas the opposite may be the case. The vibration of the foot of the fork is very slight compared with the vibration at the extremity of the prongs, and if similar intensity of vibration were used the bone conduction would be better than air. This appears a matter of very little moment, as the real use of the test is to compare air with bone conduction in normal and abnormal cases, and if a special fork is used and the result of the test on normal individuals known, then the actual question of fact as to whether bone or air conduction really is the better can be left on one side.

Twenty-five normally hearing men were examined: they were considered to have normal hearing when they could repeat accu-

rately words whispered at a distance of 16 feet. The complete result of the examinations is seen in the table. The order of the tests studied was:—

1. Distance at which a whisper was heard.
2. Weber's test.
3. Schwabach's test; the result expressed in seconds.
4. Rinne's test, right and left; the result expressed in seconds.
5. The lower tone limit; expressed by the number of vibrations producing the lowest note heard.
6. The upper tone limit, as given by the Galton-Edelmann whistle, and expressed by the number of vibrations per second which produced the highest note heard.
7. The upper tone limit, as given by the monochord; these results are only given in some of the cases on account of the objections mentioned above.

The results obtained were, on the whole, fairly uniform, particularly when one considers various factors always present to alter them. In the first place, it is impossible to get absolute quiet, and though a quiet room was used, the amount of outside noise at the time of the observation varied; again, some of the observed individuals found some of the tests difficult to give accurate answers to, particularly so with Schwabach's test; again, one must always deduct something for error in the observer—experimental error.

In *Weber's Test* the fork was not referred to either ear in any case except one, and that was doubtful.

*Schwabach's Test* gave wonderfully uniform results, except in Cases 4 and 5, where it was decidedly short, and Case 12, where it was distinctly long. The average time was 29.69 seconds, exclusive of these three cases, or 29.7, including them. More interesting is the very small deviation from the average (the average deviation is only 4.3 seconds), and in only one case (12) was the deviation more than 20 seconds, and in only two (4 and 5) was it over 10 seconds.

I think, therefore, we may consider that for the particular fork used in the aural department, the standard bone conduction

time is 30 seconds, and it may also be held that the bone conduction in normal individuals is a fairly constant amount.

*Rinné's Test* gave a positive result in all cases; in only a few cases were the two sides the same, and the amount of the excess of air conduction over bone varied considerably—from 25 seconds to 60 seconds in one case. The average for the left ear was 40.4 seconds, and for the right, 39.1 seconds. In this test the deviation from the average was, on the average, seven seconds, but there were some considerable deviations ranging from nil to 20.

However, excluding two cases, 14 and 4, the average of 40 was not departed from more than 10 seconds, and in a large number of cases not more than 6 or 7 seconds; from this we may state that Rinné's test is always positive, but that there is no fixed standard beyond the fact that the lowest amount is +25 seconds and the highest +60.

*The Lower Tone Limit.*—Almost all the cases could hear a note of 16 vibrations per second; one case (14) could only hear one of 18 per second in either ear, and in two cases (1 and 4) 16 could be heard in one ear and 18 in the other. Three cases could hear the note of 16 vibrations per second so easily that there is no doubt they could have heard one of a less number of vibrations. The conclusion to be drawn is that normally a note of 16, or at least 18, vibrations per second can be heard.

*The Upper Tone Limit*, determined by the Galton-Edelmann whistle, showed that with the exception of two cases (2 and 7), the highest note perceptible lies between one of 22,000 and 29,000 vibrations per second; the average is about 25,000, and in these two cases it was between 20,000 and 21,000. There was, as a rule, no difference between the two ears, and when it existed it was not very great.

The upper tone limit lies about a note of 25,000 vibrations per second, but varies considerably in normal individuals; the limits on the two sides is roughly the same.

The monochord was used in 10 cases; the upper tone limit given by it was, in every case, lower than that given by the whistle.

No.	Name.	Age.	Whisper audible at		Weber.	Schwabach.	Rinné.		Lower Tone Limit.		Upper Tone Limit.	
			R.	L.			R.	L.	R.	L.	R.	L.
1	G.W.	24	14ft	16ft	0	25 secs.	+28	+25	18	16	26,000	25,500
2	E.R.	24	16	16	0	25	+40	+40	16	16	20,900	20,900
3	B.F.	26	16	16	0	27	+40	+35	16	16	27,500	27,500
4	C.S.	26	16	16	0	17	+55	+53	16	18	27,500	26,500
5	G.H.	28	16	16	0	18	+	+	16	16	26,000	25,000
6	L.P.	23	16	16	0	28	+25	+30	or less	or less	26,000	27,000
7	E.V.T.	31	14	14	0	30	+40	+50	16	16	20,000	20,500
8	G.A.B.	25	16	16	0	23	+45	+45	16	16	25,000	25,000
9	H.I.D.	26	16	16	0	36	+45	+38	16?	16?	27,500	27,000
10	R.C.	30	16	16	0	32	+35	+30	16	16	27,000	27,000
11	J.L.S.	27	16	16	0	26	+40	+32	16	16	29,000	28,000
12	T.F.B.	24	16	16	0	55	+35	+50	16?	16	28,000	29,000
13	P.H.B.	23	16	16	R	34	+40	+45	or less	or less	27,000	27,000
14	R.H.L.	23	14	16	0	30	+35	+60	18	18	26,000	26,000
15	O.S.M.	23	16	16	0	30	+45	+45	16	16	24,000	24,000
16	N.G.	24	16	16	0	32	+46	+50	or less	or less	25,000	25,000
17	C.L.	22	16	16	R	25	+40	+30	16	16	23,000	23,000
18	C.H.G.	27	16	16	0	20	+37	+40	16	16	24,000	24,000
19	A.C.J.	25	16	16	0	40	+30	+40	16	15	26,000	26,000
20	W.D.S.	26	16	16	0	40	+50	+40	16	16	23,000	22,000
21	K.J.K.	23	16	16	0	26	+42	+45	16	16	26,000	26,000
22	W.R.	24	16	14	0? R.	35	+40	+40	16	16	27,000	26,000+
23	G.C.C.	24	16	16	0	40	+43	+40	or less	or less	27,000+	27,000+
24	W.E.S.D.	26	16	16	0	30	+36	+40	16	16	27,000+	27,000+
25	A.C.H.	24	16	16	0	20	+37	+40	16	16	23,000+	21,000+



## THREE CASES OF HEAD INJURY WITH DEAFNESS.

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By

W. M. MOLLISON.

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THE following three cases illustrate three different results of head injury on the sense of hearing and on the labyrinth:—

Case 1.—Charles H., aged 60, was admitted into Cornelius Ward, under the care of Mr. Lane, suffering from concussion. There was a scalp wound in the left occipital region which did not expose bone. The patient was unconscious only a few hours. He stated on recovery that he had fallen a distance of 16 feet.

*Aural condition.*—The patient, when seen the day after admission, was apparently well, but spoke in an exceedingly loud tone of voice as though deaf, and stated that he could hear nothing. On examination of the ears the tympanic membranes were found to be normal. The patient was totally deaf; no note throughout the whole range of tuning forks or Galton's whistle could be heard. Previous to the accident the hearing had been good. The patient had no vertigo, and the labyrinths were found on testing to be functionless. Both ears were syringed with cold water for several minutes; no vertigo was experienced, nor did the eyes exhibit any trace of nystagmus. Three days after admission the patient developed a cellulitis of the scalp, and a left-sided meatitis developed, which cleared up under treatment. Suppuration occurred in the left knee-joint, which was treated by drainage, and the patient made a good recovery. During all this time the patient continued to speak in a very loud voice, and all communications had to take place by writing. There was at no time any facial paralysis.

**Case 2.**—William A., aged 50, was admitted into Cornelius Ward, under Sir Alfred Fripp, for injuries to the head and arms. He had fallen a distance of 16 feet, and sustained a Colles' fracture of both wrists, and the olecranon process on the left side was fractured; there was a scalp wound in the left supra-orbital region from which fragments of bone were removed. The patient was unconscious for 24 hours.

*Aural condition.*—There was no bleeding from the ears, and the tympanic membranes were normal. There was no facial paralysis. Deafness was absolute. Previous to the accident the hearing had been good. The man is partly blind, and in any case cannot read. He complained of occasional vertigo, but his chief complaint is of headaches originating about the site of the supra-orbital wound. The labyrinths were tested by syringing with cold water (about 60° F.). This syringing produced no vertigo, but after thirty seconds nystagmus was observed; this nystagmus was of a coarse type and horizontal, best seen when the eyes were directed away from the side being tested. It lasted about one minute, and while the nystagmus was still present the patient could stand upright with feet close together and the eyes shut or open.

**Case 3.**—Henry W., aged 46, was admitted to Astley Cooper Ward in April, 1911, suffering from a fractured base of the skull. He had fallen about 15 feet on to his head, and there was a scalp wound in the left parietal region; he did not lose consciousness. There was also a fracture of the left scapula.

*Aural condition.*—There was a discharge of blood and cerebro-spinal fluid from the left ear, there was some paralysis of the left side of the face, and there was marked contra-lateral nystagmus present when the patient looked towards the right side. Three days after admission the discharge of the cerebro-spinal fluid from the ear was so copious as to soak the pillow; two days later the flow had ceased; the facial paralysis was then more marked. One week after admission a functional examination of the hearing was carried out. The right ear heard

normally, the left heard conversational speech only at a distance of six inches. The bone conduction (Schwabach's test) was normal. Rinne's test was negative on both sides; the sound was referred to the left ear from all over the head. In Weber's test the sound of the fork was referred to the left ear. The upper tone limit was normal on the right side, much depressed on the left (10,000 vibrations per second compared with 22,000 on the sound side). There was some spontaneous contra-lateral nystagmus, and there was left facial paralysis. Three weeks later the patient was again examined; the left tympanic membrane showed a scar posteriorly. He had some giddiness, but no spontaneous nystagmus was present. He heard his own voice in the left ear. The hearing had much improved; he heard whispered speech at 12 inches distance from the left ear compared with 15 feet from the right. Both Schwabach's and Weber's test were as before, but Rinne's test gave a positive result on both sides. The lower tone limit was on the right side, a note of 38 vibrations per second, and on the left, one of 55 vibrations per second. The upper tone limit was as before on the right side, but considerably higher than before on the left, being now represented by a note of 15,000 vibrations per second. The facial paralysis was less.

The first two cases are examples of total deafness following simple concussions; there was no reason to suppose a fractured base of the skull in either. In Case 1 the function of the cochlea and semicircular canals was entirely destroyed, perhaps by a hæmorrhage into them, or perhaps by damage to the nerve. It is exceedingly unlikely that the eighth nerves were both torn, because in that case the facial nerve could not have escaped. The case might, perhaps, be one of bilateral fracture of the temporal bones stopping short of the tympanum, such as was once observed by Politzer.

In Case 2 the cochlear function was entirely lost, while the vestibular function was, perhaps, almost destroyed.

Case 3 is one of fractured base of the skull, the fracture passing through the left temporal bone, probably in a direc-

tion more or less parallel to the superior border of the petrous bone. This fracture would involve the labyrinth and give rise to vertigo and nystagmus; it could involve the cochlea in part (perhaps the base only) and so give rise to the depression of the upper tone limit, and it would also damage the facial nerve lying in the aqueduct. This damage might be brought about by pressure from hæmorrhage into the canal or by pressure from slight bony displacement.

Since the upper tone limit rose during the four weeks after the accident, we may, perhaps, suppose hæmorrhage into the cochlea which gradually got absorbed.

## THE RESULTS OF OPERATIONS PERFORMED FOR THE RELIEF OF CHRONIC SUPPURATIVE OTITIS MEDIA.

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By

V. GLENDINING.

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THE object of this paper is to attempt to arrive at some conclusion as to the value of operations upon the mastoid antrum in cases of chronic otorrhoea, and the symptoms that arise as complications of long-standing inflammation in that region. In order that consecutive cases might be taken as far as was possible, it was decided to record the results of those operated upon during the year 1911. Objections may be raised as to the suitability of basing any conclusions on the conditions observed at such a short interval after the operation, the liability of these cases to relapse being so marked, but the difficulties of tracing hospital patients who were treated at any more distant period would make the series so incomplete as to be almost worthless. Even in March, 1912, when most of the present 1911 series were examined, only 19 out of 67 who were written to presented themselves for examination.

The class of patients in question is probably the most unsatisfactory possible, most of them living in extremely disadvantageous surroundings; added to this is the idea possessed by many of them that after-treatment is entirely superfluous. Most expect to be cured on discharge from the ward, a great many never even attending to be dressed. I am convinced that in many cases this led to entire failures where improvement might easily have been expected.

The 67 cases written for were only those operated on in the Aural Department, and include no cases operated on for acute complications of suppurative middle ear disease. No account is given of cases admitted for symptoms of acute mastoiditis, where antrotomies only were performed.

The procedures were, in almost all cases, the same. Where there seemed any likelihood of the ossicles being intact, and the disease limited to the mastoid antrum, a conservative operation was performed on the lines of the Kuster operation. In performing the radical operation the skin was incised along the angle between the pinna and the mastoid region, thus leaving a scar which is practically invisible except on close inspection. The soft tissues were cut through and the periosteum exposed and incised along a line directly backwards from the supra-meatal spine for the distance of an inch, and also along the posterior border of the bony meatus downwards for about the same distance. This flap was raised by a rougine and turned backwards. The cartilaginous meatus was pushed forwards and held well out of the way by a screw retractor, which held the wound open and effectually controlled most of the bleeding. The antrum was opened in the usual way, the radical operation completed by chiselling away the bridge of bone between the antrum and meatus, the tympanum curetted, and the incus and malleus removed. The cavity, cleared thoroughly of diseased tissue, was smoothed with a burr, and as much bone as was consistent with safety being removed from the facial ridge, care being taken at the same time that all parts of the cavity should be accessible from the meatus for purposes of dressing. When there was an erosion of the external semi-circular canal this was burred slightly to make it quite smooth.

A narrow knife was then passed down the cartilaginous meatus, the retractor having been removed and the tube slit up, the cut being made high up on the posterior border and reaching as far outwards as the crus helix. From this, and at right angles to it, another incision was made under cover of the inner limb of the crus, the flap thus formed being stitched with fine catgut to the soft tissues below the operation cavity, the sutures not passing through any integument. The advantage claimed for this particular method of forming the flap is that from the outside the meatus does not appear to be materially enlarged, no unsightly "cavern" presenting itself to the casual

observer. The periosteal flap was allowed to fall back into the cavity. It was usually found advisable to cut through the crus helix in an upward and backward direction far enough to allow the terminal joint of the first finger to pass from the meatus into the operation cavity. The post-auricular wound having been stitched up with interrupted horsehair sutures, a dressing of iodoform emulsion on ribbon gauze was applied to the bottom of the cavity, and as large a rubber drainage tube as possible passed down to it. Subsequently the cavity was cleaned daily with hydrogen peroxide and redressed, scarlet ointment being used as a dressing when the discharge became scanty.

In the cases where it was possible to perform the conservative operation, the procedures were exactly the same until the antrum was opened. Bone was then removed from the posterior meatal wall until a bridge about a quarter of an inch thick was left over the aditus to support the membrana tympani. The nozzle of a specially constructed syringe was introduced into the aditus from behind and air blown through it, clearing it of débris which was forced through the perforation in the membrane; following this, saline was forced through with the same syringe and the tympanum washed out as thoroughly as possible; the degree of movement of the membrane could also be observed. The same flap of meatus was utilised as in the radical operation and the same after-treatment adopted.

When no complications arose, the patients were able to leave the ward in from a week to ten days, after which they were dressed at regular intervals as out-patients, and given hydrogen peroxide (5 vols. per cent.) to drop into the cavity twice daily. One would like to insist here on the inefficiency of syringing as carried out by the friends of patients as a method of treatment; in several cases examined, although this had been carried out regularly and conscientiously, large plugs of wax were found in the meatus, effectually preventing any fluid from entering.

The following tables give briefly the main history and symptoms, together with the conditions found at operation, if worthy of note, and the result obtained:—

RADICAL OPERATIONS.

	SYMPTOMS.	OPERATION AND REMARKS.	RESULT.
1. W.B., m., <i>et.</i> 19 July 18th	Right otorrhoea, 8 months. 5 weeks post-auricular abscess, bursting and leaving sinus. Nystagmus on looking to left	A large cholesteatomatous mass had hollowed out the bone, destroying the bridge between the antrum and the tympanum, and exposing the lateral sinus and the dura mater. There was a fistula into the external semicircular canal	March, 1912, cavity quite dry. No nystagmus, no vertigo
2. G.C., f., <i>et.</i> 9 April 20th	Otorrhoea, 5 years. 3 years ago radical operation performed. 1 year ago cavity rescraped for headaches and otorrhoea	The condition of the bone suggested tuberculous disease, but this was not confirmed microscopically.	March, 1912, cavity quite dry. Headaches still persist
3. H.C., f., <i>et.</i> 23 August 23rd	Double otorrhoea for many years. August 17th, right facial paralysis. August 22nd, nystagmus, sleeplessness	Facial nerve exposed by disease for $\frac{1}{4}$ inch of its course. Fistula into external semicircular canal	March, 1912, cavity quite dry. No nystagmus, no facial paralysis
4. F.F., f., <i>et.</i> 11 June 25th	Otorrhoea for 1 year, following scarlet fever. Treatment regular	---	Cavity quite dry. Hearing improved
5. W.G., m., <i>et.</i> 7 Sept. 7th	Otorrhoea for years. February, 1910, radical operation. June, 1910, cavity rescraped. Otorrhoea persisted	Cavity found filled with fibrous tissue and spiculis of bone. More bone removed from facial ridge	March, 1912, cavity quite dry
6. R.H., m., <i>et.</i> 14 Sept. 17th	Left otorrhoea and deafness for 11 years	Since operation has been in Fever Hospital for 10 weeks	March, 1912, cavity quite dry
7. G.H., m., <i>et.</i> 43 March 19th	Left otorrhoea all his life. 15 years previously attacks of vertigo and vomiting for 1 year	After operation sent to Schiff Home	March, 1912, cavity quite dry



## RADICAL OPERATIONS—continued.

	SYMPTOMS.	OPERATION AND REMARKS.	RESULT.
8. W.H., m., æt. 10 Feb. 12th	Otorrhœa for years. Previously operated on at Victoria Hospital for acute symptoms	The disease had spread extensively backwards. 8th day following operation severe reactionary hæmorrhage	March, 1912, cavity quite dry
9. J.L., m., æt. 21	Persistent double otorrhœa for many years	Right ear: radical operation. Left ear: conservative. No after-treatment. The patient leads a healthy life as a golf caddie	March, 1912, both cavities quite dry. Hearing improved in right ear Cavity quite dry. Hearing improved
10. D.M., f., æt. 14 July 6th	Right otorrhœa and deafness for 2 years	Walls of sinus excised. Radical operation performed, but ossicles left <i>in situ</i>	Cavity quite dry. Hearing greatly improved. Sinus healed
11. M.N., f., æt. 20 July 19th	Right operation 13 years ago for acute symptoms with success. Otorrhœa for a few months in 1910. March, 1911, post-auricular abscess leaving sinus	Lateral sinus and dura mater exposed	Cavity quite dry
12. C.O'C., m., æt. 83 June 30th	Right otorrhœa for 10 years. 3 weeks' pain	—	March, 1912, cavity quite dry
13. J.P., m., æt. 11 March 31st	Left otorrhœa for years	Suppuration in the wound followed	Cavity quite dry
14. A.R., m., æt. 15 Sept. 28th	Right otorrhœa for years. Two previous operations 4 and 1 years ago without success	At the operation the facial nerve for $\frac{1}{4}$ of its course was accidentally exposed, leading to facial paralysis. The external semicircular canal was also opened	Cavity quite dry. No facial paralysis, no nystagmus, no vertigo
15. G.N., m., æt. 22 October 8th	Left otorrhœa 3 years. Vertigo during last year		

RADICAL OPERATIONS—continued.

	SYMPTOMS.	OPERATION AND REMARKS.	RESULT.
16. A.W., f., æt. 32	Persistent otorrhœa	—	Cavity quite dry
17. V.F., f., æt. 10 August 13th	In 1908 the patient was in Job Ward and was operated on for lateral sinus thrombosis; the jugular vein was ligatured and the wound drained behind the ear. On admission there was a sinus behind the ear with a very foul discharge. No nystagmus or vertigo	The walls of the sinus were excised and the radical operation completed; considerable areas of dura mater were exposed. Fistula in external semicircular canal	March, 1912, post-auricular wound healed perfectly. Very slight thin discharge from cavity
18. F.H., f., æt. 10 May 18th	Left otorrhœa for several years. Regular treatment for 7 months without improvement	—	Much improved. Cavity quite dry, but some pus in tympanum
19. W.H., m., æt. 19 July 6th	Many years otorrhœa. May, 1910, Radical operation without success	Some necrosed bone removed from facial ridge	Improved. Not quite dry. Treatment with $H_2O_2$ drops caused giddiness, so was not persisted in
20. M.H., f., æt. 11 May 25th	Left otorrhœa for 3 years following measles	—	Improved, but cavity not quite dry
21. A.K., m., æt. 66 June 27th	Right otorrhœa 6 years. Attacks of deafness, giddiness and vomiting for 12 months. 14 days previous to admission marked vertigo and vomiting, falling in any direction. Nystagmus to left	Large cholesteatomatous mass found. Fistula in external semicircular canal. Lateral sinus and dura mater exposed. Wound drained behind as well as in front	Post-auricular wound healed. Not much discharge of a thin fluid; no pus. Slight nystagmus. Vertigo unimproved

## RADICAL OPERATIONS—continued

	SYMPTOMS.	OPERATION AND REMARKS.	RESULT.
22. E. F., f., æt. 23 June 11th	Double otorrhœa 12 years. Progressive deafness 4 years. Double mastoid operation 4 years ago. Pain in left ear and vertigo previous to admission	Left side reopened and scraped	July, 1911, cavity quite dry. Commenced to discharge again after a bad throat in March, 1912. Unhealed area at the top of the cavity was in a healthy condition. Hearing improved. No vertigo
23. R. V., m., æt. 32 June 11th	Left otorrhœa and almost complete deafness since childhood. 3 weeks previous to admission pain, with attacks of vertigo, falling to right	Large amount of cholesteatoma. Fistula into external semicircular canal	Practically healed except for a very small area at the top of the cavity. No vertigo
24. W. T., m., æt. 15 June 17th	Right-sided otorrhœa since infancy. No vertigo, no nystagmus	Radical operation. Fistula in external semicircular canal. Dura mater of middle fossa exposed and bulged down, making the clearing of the cavity almost impossible	Improved. Pus in tympanum. Cavity almost completely covered with epithelium
25. T. C., m., æt. 8 Nov. 18th	Right-sided otorrhœa since infancy. 2 years ago antrotomy for acute symptoms. Discharge continued. Tenderness 3 weeks. No vertigo, vomiting nor nystagmus	Bone very soft and porous. Cholesteatoma present. Fistula in external semicircular canal	Still discharging freely. Has never attended since leaving the ward
26. C. C., m., æt. 20 August 10th	Otorrhœa for 10 years. 1908, conservative operation performed with good result. Otorrhœa recommenced Sept., 1909. Attacks of vertigo and vomiting for some months	Radical operation completed	The cavity still discharges freely. No vertigo nor vomiting

RADICAL OPERATIONS—continued.

	SYMPTOMS.	OPERATION AND REMARKS.	RESULT.
27. A.C.N., m., æt. 11 August 29th	Right otorrhœa 2 years. 3 weeks' history of deafness, drowsiness, constipation, and enlargement of the abdomen. When roused the patient could answer fairly intelligently. Very ill when transferred from Stephen Ward. Tenderness and swelling behind right ear. No optic neuritis	Very extensive disease. Lateral sinus and dura mater over both cranial fossæ exposed, but found healthy and pulsating. Condition not improved. September 6th dura mater of middle fossa incised and a gauze drain inserted, allowing escape of cerebro-spinal fluid for 2 days. Condition improved. Dr. Hertz diagnosed tubercular peritonitis. Sent to Schiff H. me	March, 1912, the cavity was still discharging. General condition much improved and patient in fairly good general health
28. J.R., m., æt. 13 Feb. 8th	Right otorrhœa for years	—	Still discharging freely
29. E.S., f., æt. 7 April 5th	Otorrhœa for 6½ years	—	Still discharging. Has not attended since leaving the ward
30. A.S., f., æt. 21 August 17th	Right otorrhœa 5 years. 1909, An-trotomy for acute symptoms. Lately has suffered from attacks of vertigo, falling to right, and nausea. No nystagmus	The cavity was skin grafted	Still discharging. Has not attended since leaving the ward. No vertigo
31. D.S., m., æt. 11 Feb. 5th	Double otorrhœa of long standing	Left ear—radical operation. Right ear—conservative operation.	Both still discharging. Left cleaner than right
32. F.S., f., æt. 31 July 18th	Persistent double otorrhœa	Radical operation on both sides	Both ears still discharging

## CONSERVATIVE OPERATIONS.

	SYMPTOMS.	OPERATION AND REMARKS.	RESULT.
33. B.A., f., æt. 19 May 2nd	Right otorrhœa intermittently since childhood. Attacks of vertigo (2 weekly) "for years"; objects move from left to right; requires support to prevent falling during attack, which can be brought on by looking to the left. Slight nystagmus to left.	No injury to semicircular canals. Conservative operation	Cavity quite dry. Only occasional slight giddiness. Hearing improved
34. M.O., f., æt. 19 Jan. 1st	Right otorrhœa 4 years, with occasional pain	Conservative operation	Dried up 12 weeks after operation, but relapsed. March, 1912, quite dry. Hearing improved
35. J.H., m., æt. 12 Jan. 28th	Otorrhœa for years. Occasional giddiness	Conservative operation	Quite dry. Hearing improved
36. F.O., m., æt. 13 August 18th	Left otorrhœa 9 months. Pain 1 month, with giddiness and nausea	Conservative operation. Dura mater exposed, lateral sinus torn	Quite dry. Hearing not improved. No giddiness
37. L.R., f., æt. 26 Feb. 2nd	Left otorrhœa 5 years	Conservative operation, followed on 3rd day by sepsis in wound	Quite dry. Hearing improved
38. J.R., m., æt. 22 March 9th	Right otorrhœa since childhood. Worse for last 2 years	Conservative operation. Cavity very foul. Lateral sinus exposed	Still discharging. Left-sided nasal obstruction. Tonsils unhealthy
39. K.M., f., æt. 6 Jan. 12th	Left otorrhœa and deafness since infancy	Conservative operation	Quite dry. Hearing had improved, but has relapsed into former condition

Of the first series of 32 cases on whom the radical operation was performed, in one case radical operations on both sides were completed, and in two cases a radical operation on one side and a conservative operation on the other, thus bringing the total of operations up to 35; of these, on inspection in March, 1912, 17 cavities were found to be quite healed, and no signs of discharge present (one being a conservative), 8 were markedly improved and showed signs of healing in the near future; of the remaining 10 (one conservative included), although some were undoubtedly improved to a considerable extent, others showed no signs of the discharge lessening; in three of these cases the patient had not been dressed since leaving the ward, so that little success could be hoped for in their case. In five cases the hearing had improved.

In the series of conservative operations, nine altogether (two being included from the first table, performed on one side, the radical operation being completed on the other), seven were perfectly dry, two were unimproved; in one of these, nasal obstruction and unhealthy tonsils had been overlooked. In four of these patients the hearing had also improved greatly.

In certain cases the operation was performed because of symptoms or signs indicating involvement of the neighbouring structures; of the eight such cases who had suffered from attacks of vertigo or vomiting, six were entirely relieved of the symptoms; in one case the frequency and severity of the attacks was greatly diminished, and in the remaining case no improvement followed. In the last two cases nystagmus was also present and unrelieved by the operation; two other cases of nystagmus were, however, relieved entirely. In one case of severe and persistent headaches, otorrhoea was the only sign that could be found to account for the symptoms, but the cavity healed perfectly, and yet no benefit to the headaches was derived. One case, operated upon because of facial paralysis, which had developed a few days previously recovered completely.

Of 12 patients who exhibited signs of labyrinthine disease, in five a fistula was found to exist between the external semicir-

cular canal and the antrum, whilst in three cases the fistula was present without giving rise to any symptoms which would lead one to suspect its presence.

In collecting this series of cases, one was struck by the larger proportion of successes following the conservative operation. This can easily be accounted for by the more limited extent of disease which permitted this operation and the more healthy state of the tympanum. In nearly all cases this portion of the cavity was the last to heal, owing to the difficulty of clearing it thoroughly of all diseased tissue without dislocating the stapes from the fenestra ovalis.

The hope that one can hold out to patients of relieving such distressing symptoms as vertigo, vomiting, etc., seems to quite justify the operation in these cases. With regard to hearing, a marked improvement usually followed the conservative operation, but it is extremely doubtful if any of those who are now improved by the radical operation will remain so, many stating that they could hear much better for some time after the operation, but had since relapsed into their former condition; none complained of their hearing being worse.

In summing up, it would seem that the results obtained are much more satisfactory than one expects in this class of patient. Of the 42 operations, 23 were entirely successful in stopping the otorrhoea, 8 cases showed signs of healing and of soon becoming quite dry, whilst 11 were still discharging, and in only six of these (Nos. 24, 28, 29, 30, 31, 37) can it be said that the patient derived either no local or general benefit from the operation; of these, three had had no after-treatment, and in one, nasal obstruction and unhealthy tonsils had been overlooked, thus leaving two cases that had entirely failed to improve under fair conditions.

In conclusion, it must be insisted upon that all these cases, previous to operation, had received regular and systematic treatment for their condition at Out-Patients.

# OBSERVATIONS ON THE TEMPERATURE OF MAN AFTER TRAUMATIC SECTION OF THE SPINAL CORD.

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By

H. GARDINER, M.B., B.S.,

AND

M. S. PEMBREY, M.A., M.D.

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INTRODUCTION.

CLASSIFICATION OF CASES—

(i.) According to Temperature.

Hyperthermia.

Hypothermia.

(ii.) According to Site of Lesion.

Cervical.

Dorsal.

Lumbar.

CONCLUSIONS.

REFERENCES.

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INTRODUCTION.

It is now a hundred years since Benjamin Brodie<sup>1</sup> first drew attention to the influence of the nervous system upon the temperature of the body. He divided the spinal cord of a mammal



high up in the cervical region and found that the circulation of the blood continued if artificial respiration was performed, but the temperature of the animal fell rapidly. In a man whose spinal cord had been crushed at the level of the fifth and sixth cervical vertebræ he observed that the temperature rose to  $43.9^{\circ}$  ( $111^{\circ}$  F.) before death, which occurred twenty-four hours after the accident. No satisfactory explanation could be advanced for such results; the knowledge of animal heat was too incomplete at that time.

The observations of Benjamin Brodie led to numerous experiments and discussions on the subject, but no agreement could be obtained in either the experimental or clinical data. In 1878 Pflüger<sup>2</sup> published experiments upon the respiratory exchange of rabbits, and showed that the animal after section of the spinal cord in the cervical region is comparable to a cold-blooded animal; a rise in external temperature increases, a fall diminishes, the production of carbon dioxide and heat. Herein, it would appear, lies the clue required to explain the discordant results.

Pflüger's work, however, did not produce much effect upon the clinical opinion of that time, and even at present physicians and surgeons appear to be satisfied with the more modern theories of special "heat centres" in the brain. An explanation based upon such debatable centres is of extremely doubtful value. The existence of special "heat centres" is unproven, but it is true that by suitable modifications the theory can be made to explain the facts which have been observed.

Several years ago one<sup>3</sup> of us advanced an explanation based upon experiments along the lines introduced by Pflüger, and supported by observations upon the temperature of animals<sup>4</sup> and man after section of the spinal cord. Since that time further experiments and observations have been made; they confirm the earlier conclusions which we now quote. "The general result is a subnormal temperature so long as the patient's condition is

not complicated by other internal or external disturbance. The subnormal temperatures are due to excessive loss and diminished production of heat owing to the vaso-motor and motor paralysis. The section of the spinal cord high up in the cervical region abolishes the power of regulating temperature. When the patient is exposed, even to moderate cold, his temperature falls owing to the increased loss of heat and to the diminished production of heat. On the other hand, if the weather be hot and the patient too well covered with bed-clothes, his temperature rises and may reach a dangerous height owing to the diminished loss and the increased production of heat in the body. In the paralysed man the production of heat rises and falls *with* the external temperature. In the case of the high temperatures there are several factors which may play an important part; the paralysed parts soon cease to sweat; in fact, Horsley has shown that, by the use of pilocarpine, it is possible to localise the level of the injury to the cord. The respiration is hampered, it is only diaphragmatic; the ventilation of the lungs is, therefore, imperfect, and less heat is lost by the cooling of the inspired air and by the evaporation of water from the respiratory tract to saturate the expired air with moisture. Further, the warmer the paralysed tissues the greater is their metabolism and production of heat. It naturally follows that, in case of section of the spinal cord in the dorsal or lumbar region, the regulation of temperature is less disturbed."

A further discussion, however, will be postponed until the details of the records of the cases have been given.

#### CLASSIFICATION OF CASES.

The present paper deals with all the cases which we could find recorded in the clinical reports of Guy's Hospital, and covers a period of nearly fifty years. Some of the reports are too imperfect to be of any real value; in other cases there were complications at the time of the accident, such as fracture of the skull, and in another large group there were complications aris-

ing after the accident, such as cystitis, bed-sores, and bronchopneumonia. More than a hundred reports have been examined. After rejection of 25 cases owing to incompleteness, there remain 78 cases for the purpose of classification. It is possible to adopt either a physiological or an anatomical basis for the classification. Both are given below. For the purpose of explanation the physiological is to be preferred, but for complicated cases the anatomical is more convenient.

The temperature of the patient may (i.) rise above the normal—hyperthermia, (ii.) fall below the normal—hypothermia, or (iii.) remain at the normal level, although the capacity for regulation is impaired. In the following tables of hyperthermia and hypothermia we give, with one exception, only the uncomplicated cases, and every record of temperature is included in the tables.

## HYPERTHERMIA.

No. of Case.	Temperature.	Pulse.	Respiration.	Level of Lesion.	Interval between Accident and Death in Days.	REMARKS.	Year.	Surgeon.	No. of Clinical Report.
5	99.4°	—	—	5, 6 C.	3	Skin dry	1909	Mr. Dunn	571
	106 sponging	—	—	—	—				
	100.8	—	—	—	—				
	101.4	—	—	—	—				
	99.4	—	—	—	—				
	104.0	—	—	—	—				
32	102.4	—	—	—	—	Marked sweating on face and neck, but none on limbs	1890	Mr. Durham	346 A.
	105.4 ax. 107	116	42	5, 6 C.	1				
33	98	60	20	5, 6 C.	1	Post-mortem	1890	Mr. Lucas	409
	97.4	—	—	—	—				
	102.6	—	—	—	—				
	104	120	—	—	—				
	107.6	90	24	—	—				
40	105	84	—	5, 6 C.	1	Post-mortem	1883	Mr. Howse	307
	107.6 ax. (R)	—	—	—	—				
	107.8 ax. (L)	—	—	—	—				
	sponging	—	—	—	—				
	107.8	—	—	—	—				
45	106.3	—	—	—	—	Post-mortem	1882	Mr. Howse	288
	96.8	—	—	5, 6 C.	1½				
	96	144	—	—	—				
	96	172	—	—	—				
	107.6	—	—	—	—				

## HYPERTHERMIA—continued.

No. of Case.	Temperature.	Pulse.	Respiration.	Level of Lesion.	Interval between Accident and Death in Days.	REMARKS.	Year.	Surgeon.	No. of Clinical Report.
55	100.4° 102.2 106 109	70 102 130 170	— — — —	5, 6 C.	1 — — —		1881	Mr. Durham	294
59	103 107.6 109.3 109.2	100 146 — —	— 36 — —	4, 5 C.	1 — — —		1879	Mr. C. Forster	344
61	104 104.4 106.8	— — 114	— — 48	4, 5, 6 C.	3 — —	Sweating	1879	Mr. C. Forster	346
64	95 103 103 106 107.8 cold douche	64 108 — 120 132	— — — — 32	6 C.	2 — — — —		1878	Mr. Howse	228
69	105 106 106 106.8	120 120 108 —	36 — 32 —	— — — 6 C.	— — — 1		1878	Mr. Bryant	302

## HYPERTHERMIA—continued.

No. of Case.	Temperature.	Pulse.	Respiration.	Level of Lesion.	Interval between Accident and Death in Days.	REMARKS.	Year.	Surgeon.	No. of Clinical Report.
73	102.2° 102 102 103 103 102.6 102 102 102 102.6 102.6 100.5 100.8 102.6 102 103.4 105 105.2 107.2	96 94 96 88 92 92 80 80 84 94 80 80 92 100 100 — — —	— — 34 25 22 28 26 24 27 28 28 28 28 28 — — — — —	5, 7 C.	4		1876	Mr. Howse	242
74	101.2 105.5 106.4 106.8 107 108	60 100 96 96 —	16 38 48 42 —	2 D.	2	One hour after death	1876	Mr. Durham	272

## HYPERTHERMIA—continued.

No. of Case.	Temperature.	Pulse.	Respiration.	Level of Lesion.	Interval between Accident and Death in Days.	REMARKS.	Year.	Surgeon.	No. of Clinical Report.
76	96.8° 104 107.5 108.2 108.4 108.4 108	— 84 112 112 124 — 122	— 40 44 — — — 36	C. — — — — — —	1 — — — — — —		1875	Mr. Howse	232
78	105 105	118 118	24 24	6 C. —	1 —	Convulsions. Skin hot and dry	1875	Mr. C. Forster	315
81	103.2 104.6 105.8 108	87 — — —	24 — 34 —	6 C. — — —	1 — — —	Post-mortem	1874	Mr. C. Forster	327
84	102.2 104.4 109	96 94 116	— — 42	7 C., 1 D. — —	2 — —		1873	Mr. Durham Mr. Davies-Colley	206 A.
92	103.6 105.2 104.2 103 106.6 105.4 106 103 104.6 104	84 84 78 72 90 72 78 66 78 80	— 36 28 26 32 28 35 27 36 36	5, 6, 7 C. — — — — — — — — —	4 — — — — — — — — —		1871	Mr. Bryant	440





## HYPOTHERMIA.

No. of Case.	Temperature.	Pulse.	Respiration.	Level of Lesion.	Interval between Accident and Death in Days.	REMARKS.	Year.	Surgeon.	No. of Clinical Report.
11	Below 90° 95·6 97·2	48 — 76	— — —	7 C. — —	1 — —	Bronchitis and fracture of manubrium sterni	1906	Mr. Golding-Bird	519
15	95·2	—	—	4, 5 C.	$\frac{1}{2}$		1903	Mr. Symonds	175
18	95 96·8	— —	— —	4 C. —	1 —		1895	Mr. Lucas	468
86	97 95	— 76	— —	6, 7 C. —	2 —		1873	Mr. C. Forster	327
88	97 ax. 96	60 66	15 39	5, 6 C. —	3 —		1872	Mr. C. Forster	355
90	97·6	—	—	3, 4 C.	$\frac{1}{2}$		1871	Mr. C. Forster	322

In a discussion of these cases it is necessary, in the first place, to point out that the records of the temperature have not the full scientific value which they would have if they had been taken with more care. The figures are probably too low in the case of the high temperatures, and too high in the case of the very low temperatures, for the following reasons. The observation of the temperature in the mouth is unreliable, especially when the patient is breathing with the mouth open or suffering from dyspnoea. The ordinary clinical thermometers are graduated only as low as 95°, and records below that figure are entered as "95°" or "below 95°." It is unfortunate that there is prevalent in most hospitals the idea that the observation of the temperature of a patient is so simple that it can be left to the nurse, and that no special instruction upon the subject is necessary for the medical student. The result is that many points of extreme interest in the physiology and pathology of animal heat and fever are unsolved, although the material has been available.

An examination of the two tables already given shows that there are 19 uncomplicated cases of *hyperthermia*. The average duration of life after the accident was two days, and the highest temperature recorded was 109.2° (42.9° C.). With one exception the lesions were in the cervical region. The records of the rate of respiration often show an increase corresponding to the rise in temperature, but it is noteworthy that the pulse in many cases exhibits no such change. Sweating was observed on the sensitive parts, but not on the paralysed portions of the body.

In the records of *hypothermia* there are five cases without complications. The lowest temperature is given as "below 90°." The average duration of life after the accident was about a day and a half, and the lesion in each case was in the cervical region. Very low temperatures would not be expected in patients placed in warm beds after the accident and living only for a short time. Lower temperatures are given in cases in which life lasted for a longer time; for example, a chart is given

later of a temperature below  $80^{\circ}$  ( $26.7^{\circ}$  C.) for two or three days. In the uncomplicated cases of hypothermia there is no doubt that the cause of death was not the low temperature, for observations prove that much lower temperatures are not incompatible with life. The range of safety below the normal is almost twice as great as that above the normal temperature. The low temperature must be considered as one of the signs of the reduced vitality of the patient after the accident, and death may have been due to failure of the circulation owing to the loss of vaso-motor control over the greater part of the body.

## COMPLICATED CASES—CERVICAL REGION.

No. of Case.	Temperature.	Pulse.	Respiration.	Level of Lesion.	Interval between Accident and Death in Days.	REMARKS.	Year.	Surgeon.	No. of Clinical Report.
1	95 to 99°	—	—	7 C & 1 D	5	Cystitis	1910	Mr. Dunn	168
2	96.2 to 108	—	—	5	4	Fracture of sternum. Slight hemorrhage into lungs, and posterior mediastinum and under pia mater	1909	Mr. Symonds	408
3	96.8 to 101.4	—	—	6, 7	5	Recent right apical lobar pneumonia. Extravasated blood in deep tissues behind fractured sternum	1909	Mr. Symonds	443
7	95 to 98.4	—	—	6, 7	3	Pleurisy, bronchitis, delirium, coma	1908	Mr. Dunn	524
12	97 to 103.6	—	—	5, 6	4	Septic broncho-pneumonia	1905	Mr. Symonds	28
13	95 to 102.8	—	—	5, 6	177	Cystitis. Bed sores	1904	Mr. Lucas	210
20	99.4 to 103.4	—	—	5	10	Pleurisy. Broncho-pneumonia	1895	Mr. Lucas	470
23	95 to 100.6	—	—	7	29	Broncho-pneumonia. Ascending nephritis	1893	Mr. Davies-Colley	421
24	97 to 102.6	—	—	7	141	Laminectomy. Bed sores. Broncho-pneumonia	1893	Mr. Davies-Colley	477

## COMPLICATED CASES—CERVICAL REGION—continued.

No. of Case.	Temperature.	Pulse.	Respiration.	Level of Lesion.	Interval between Accident and Death in Days.	REMARKS.	Year.	Surgeon.	No. of Clinical Report.
28	98.6 to 105°	—	—	3, 4	8	Laminectomy. Pneumonia	1891	Mr. Durham	360
36	98 to 99	—	—	6, 7	4	Bed sores	1888	Mr. Howse	258
42	97.8 to 100.9	—	—	6	3	Edema of lungs. Fatty heart	1883	Mr. Howse	285
43	Below 95 to 103	—	—	7	11	Pneumonia. Cystitis	1882	Mr. Howse	286
45	101	—	—	5, 6	1	Lungs congested. Respiratory failure	1882	Mr. Howse	289
47	97.2 to 104	—	—	7	193	Cystitis, pyelitis, lardaceous disease. Sweated freely on sensitive parts; non-sensitive parts dry.	1882	Mr. Durham	298
48	96 to 105.8	—	—	5	47	Cystitis, bed sores, early broncho-pneumonia	1882	Mr. Bryant	278
49	90.1 to 108.6	—	—	5	527	Cystitis, suppurating kidneys, lardaceous viscera. Low temperature in mouth during rigor	1883	Mr. Bryant	279
50	95 to 101.2	—	—	5, 6	2	Fractured sternum, lungs congested	1881	Mr. Davies-Colley	298

## COMPLICATED CASES—CERVICAL REGION—continued.

No. of Case.	Temperature.	Pulse.	Respiration.	Level of Lesion.	Interval between Accident and Death in Days.	REMARKS.	Year.	Surgeon.	No. of Clinical Report.
56	100 to 108°	—	—	5, 6	17	Cystitis	1880	Mr. Durham	266
57	102 to 102·2	—	—	4, 5?	2	Cystitis	1880	Mr. Bryant	325
58	99·4 to 100·4	—	—	5	1	Dyspnoea. Accumulation of mucus in trachea and bronchi	1880	Mr. Bryant	326
63	97·6 to 103·8	—	—	6, 7	14	Fractured jaw. Pneumonia. Cystitis. Suppurating kidneys.	1879	Mr. C. Forster	348
67	96·4 to 103	—	—	?	31	Scalp wounds. Recto-vesical fistula. Bed sores	1878	Mr. Howse	241
68	99·6 to 106	—	—	5	13	Slight emphysema and oedema of bases of lungs	1878	Mr. Bryant	301
80	98·2 to 105	—	—	5, 6	4	Fracture of 4, 5, 6 ribs. Oedema of lungs	1875	Mr. C. Forster	317
89	103·4	—	—	5, 6	3	Cystitis	1872	Mr. Bryant	380
93	95·8 to 102·7	—	—	6, 7	5	Cystitis. Lungs congested	1870	Mr. Cook	297
94	99 to 104·6	—	—	5, 6, 7	3	Broncho-pneumonia. Miliary tubercle	1870	Mr. Poland	328

## COMPLICATED CASES.—DORSAL REGION.

No. of Case.	Temperature.	Pulse.	Respiration.	Level of Lesion.	Interval between Accident and Death in Days.	REMARKS.	Year.	Surgeon.	No. of Clinical Report.
9	Below 95° to 104·6	—	—	3	12	Softening and small hemorrhage in cord, broncho-pneumonia, suppurative nephritis of both kidneys	1907	Mr. Lane	604
16	103 to 104	—	—	6	12	Acute pleurisy, pyelonephritis	1896	Mr. Davies-Colley	362
17	96·4 to 104	—	—	Lower	330	Cystitis	1895	Mr. Golding-Bird	429
21	97 to 105	—	—	5, 6	13	Laminectomy, sepsis	1894	Mr. Golding-Bird	42
22	96·2 to 104·2	—	—	4, 5	56	Broncho-pneumonia, bronchitis, cystitis	1894	Mr. Golding-Bird	366
25	96 to 105	—	—	Middle	1275	Laminectomy, bedsores, broncho-pneumonia	1892	Mr. Davies-Colley	352
30	99 to 102·4	—	—	4, 5	3	Fractured patella and sternum, granular kidneys	1891	Mr. Lucas	374
51	98 to 99	—	—	11, 12	2	Cystitis, pyelonephritis	1881	Mr. Davies-Colley	299
52	98 to 99	—	—	10, 11	14	Cystitis, polyuria, pyelitis	1881	Mr. Davies-Colley	300
54	99·1 to 101·7	—	—	10	8	Cystitis, pyelonephritis	1881	Mr. Howse	299
60	103 to 106·8	—	—	2	24	Cystitis, bedsores	1879	Mr. C. Forster	345
79	101·4	—	—	Upper	1	Fractured ribs, right femur and patella. Sweating above 4th rib, skin dry below	1875	Mr. C. Forster	316
83	97 to 103·3	—	—	12	20	Extravasation of urine, spinal cord pulped at seat of injury	1874	Mr. C. Forster	329
87	99 to 103	—	—	5, 6	228	Lardaceous viscera	1873	Mr. Poland	243

## COMPLICATED CASES—LUMBAR REGION.

No. of Case.	Temperature.	Pulse.	Respiration.	Level of Lesion.	Interval between Accident and Death in Days.	REMARKS.	Year.	Surgeon.	No. of Clinical Report.
98	98·4° to 101·8	—	—	2	8	Pneumonia, pleurisy, fracture of 1st and 4th ribs	1890	Mr. Lucas	422
99	98 to 100	—	—	4 or 5?	Discharged after 7½ mths.	Complete paraplegia at first. Discharged using crutches	1891	Mr. Davies-Colley	335
37	98 to 101	—	—	2, 3	33	Septic hæmatoma, thrombosis of left iliac veins, delirium	1888	Mr. Bryant	123
70	97 to 101·2	—	—	2, 3	20	Cystitis	1877	Mr. Durham	254
102	97 to 98·8	—	—	1, 2	Discharged after 28 days		1884	Mr. Davies-Colley	282
103	97 to 102·6	—	—	1, 2	Discharged after 32 mths.	Cystitis. Discharged using crutches	1882 1883	Mr. Durham Mr. Durham	300 312
104	97 to 98	—	—	?	Discharged after 4 mths.	Cystitis	1881	Mr. Davies-Colley	301
75	96 to 105	—	—	1	22	Cystitis, suppurating kidneys	1876	Mr. Bryant	307



Among the complicated cases are 28 cervical, 14 dorsal, and 8 lumbar lesions; the highest temperature in each series was 108° (42.2° C.), 106.8° (41.5° C.), and 105° (40.5° C.) respectively. If the uncomplicated and the complicated cases are considered together the range of temperature observed is as follows :—

Maximum.	Minimum.	Range.	Number of Cases.
109.2° (42.9° C)	77.7° (25.4°)	31.5° (17.5°)	55 Cervical
108 (42.2 )	Below 95 (35 )	13+ ( 7+ )	15 Dorsal
105 (40.5 )	96 (35.5 )	9 ( 5 )	8 Lumbar

A comparison of the average maximum temperature in the dorsal and lumbar cases indicates that similar complications, such as cystitis, pyelo-nephritis, and bed-sores, cause the temperature to rise to a level about 2° higher in the dorsal than in the lumbar cases. This, taken in conjunction with the above table of the range of temperature, supports the view that the regulation of heat in a paraplegic patient is effected through the unparalysed parts of the body, and, provided that the body is kept warm, the active means of lowering the temperature vary inversely as the area of paralysis.

In the normal body the regulation of the loss and the production of heat depends upon the integrity of the central nervous system, but in the cases which we are considering this integrity has been broken, and the body may be regarded as one divided into two portions: one, the non-paralysed, in which the regulation is still active, and the other, the paraplegic, in which the regulation has ceased. It therefore follows that the greater the extent of the paralysis the greater the strain which will be thrown upon the unparalysed parts, and there will come a time when the latter, in the case of a high lesion, will be quite incapable of keeping the temperature of the whole body within safe limits. As, therefore, it would be expected, the temperatures given above show that the widest variation from the normal is found in the case of lesions in the cervical region, and the least in those affecting the lumbar region.

In a paraplegic animal experiments show that the increased loss and the diminished production of heat in the paralysed parts may be compensated by (i.) more active production and diminished loss in the non-paralysed parts, or (ii.) warm surroundings. In man the latter method is the one adopted as a routine, and consequently there are far fewer cases of hypothermia than might be expected from the results of experiments upon animals. Another reason is the difference in the relation of the surface of the body to its mass; a small animal is much more quickly cooled than a large one. There remains another important cause of hyperthermia: the paralysed portions of the body produce more heat as their temperature is raised, and thus a so-called vicious circle is established. The initial rise of temperature may be due to external warmth or infection; in the former case a condition of *hyperthermia* would arise as distinguished from one of *hyperpyrexia* in the latter case.

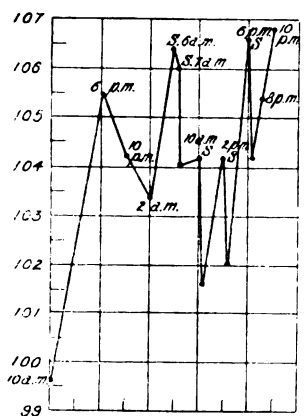


FIG. 1.

Case No. 27. Chart for August 11th and 12th, 1891. Level of lesion 5th cervical vertebra. Post-mortem examination showed one patch of bronchopneumonia. S=sponging.

If the explanation which has been advanced is correct, it would follow that a high temperature could be reduced by a sufficient application of cold, and a low temperature raised by artificial warmth. This has been proved both for man and animals after

section of the spinal cord, and in this connection the following charts of three special cases, which have not been given in the tables, are of interest. In Case No. 27 (Fig. 1) there is a note in the report for August 11th that "patient's breathing has become somewhat laboured, and it was thought that he had developed pneumonia; his temperature has gone up to 105°; he has been put into a warmer bed, well out of draughts." The temperature chart shows the subsequent course of events; by sponging, the temperature was not reduced below 101°, and rose again to 106·8°, when the patient died. As a contrast to this may be given Case No. 8 (Fig. 2). The temperature

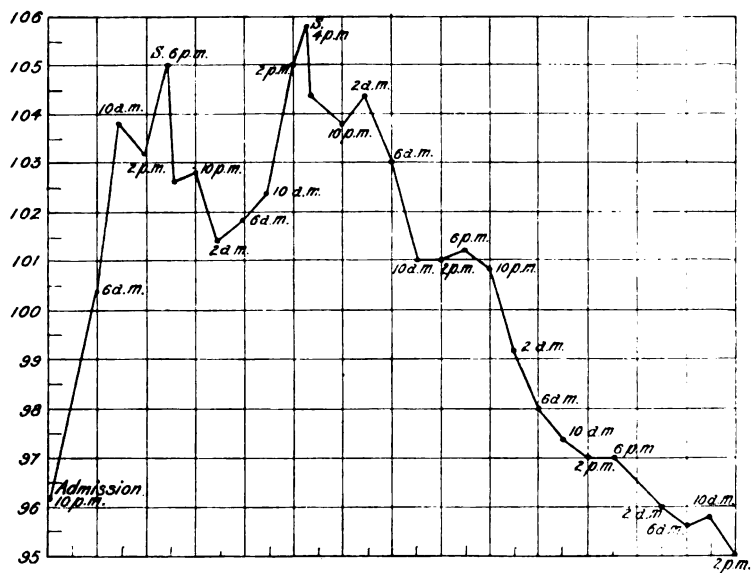


FIG. 2.

Case No. 8. Chart from June 1st to June 6th. Level of lesion about 4th cervical vertebra; post-mortem data incomplete. S=sponging.

reached a height of 105·8°, but was reduced by sponging, and fell within two days to 97°, continued to fall during the third day to 95°, and remained at 96°, or "below 95°" until death 16½ days later. In the third Case, No. 34 (Fig. 3), the temperature steadily fell to 77·7° in 25 days, the interval between the accident and death.

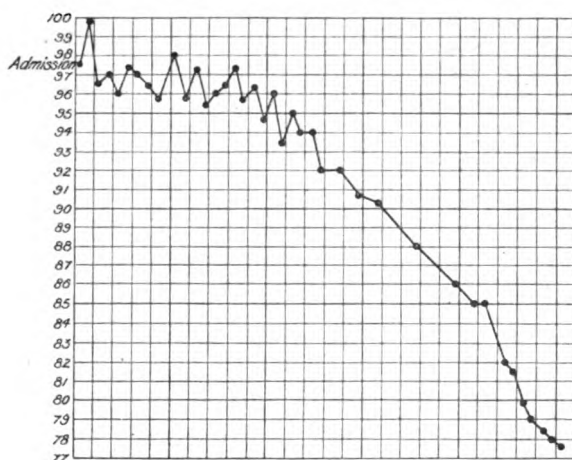


FIG. 3.

Case No. 34. Chart from December 9th, 1888, to January 2nd, 1889. Post-mortem showed fracture between 6th and 7th cervical vertebræ, bronchopneumonia and cystitis. 1 day=2 divisions.

There are further points of interest to be considered. The average length of life after the accident was, for the cervical lesions 23.6 days, for the dorsal, 133 days, and for the lumbar, 176(+). In the last cases no definite figure can be given, for several of the patients were discharged from the hospital.

The complications which followed the accidents show, as would be expected, a relationship to the seat of lesion, but the incidence must be considered in relation to the duration of life after the accident.

There are many points which require further investigation, such as the blood pressure, the pulse, rigors, and the influence of infection upon the regulation of the temperature already impaired by the lesion of the spinal cord. Many of these questions could be studied more readily upon man than upon animals, but the opportunities cannot be foreseen.

*Observations on the Temperature of Man  
after Traumatic Section of the Spinal Cord.*

Total No. of Cases.	No. of Complicated Cases.	Region of Lesion.	Cystitis,			Broncho-Pneumonia.			Average duration of life after accident.	
			Per cent. of			Per cent. of			Total Cases. Days.	Complicated Cases. Days.
			Total No.	Total Cases.	Complicated Cases.	Total No.	Total Cases.	Complicated Cases.		
55	29	Cervical	13	23·2	44·8	13	23·2	44·8	23·6	41·2
15	14	Dorsal	8	53·3	57·1	3	20	21·4	133	142
8	7	Lumbar	4	50	57·1	1	12·5	14·3	176 (+)	195·4 (+)

### CONCLUSIONS.

The temperature of man after traumatic section of the spinal cord may rise above the normal, *hyperthermia*, fall below the normal, *hypothermia*, or remain at the normal level, although the capacity for regulation is impaired. In a paraplegic patient the body is divided into two portions: the non-paralysed and the paralysed; in the former the capacity to regulate temperature is present, in the latter it is absent; only within narrow limits can the former compensate for the latter by increased production or loss of heat, as the case may require. The site of the lesion will determine the relative proportions of the two parts, and for this reason cases of section of the spinal cord in the cervical region will show the greatest variations from the normal temperature.

In the paralysed portion of the body there is no control over the production and loss of heat; the production rises and falls *with* the temperature, and sweating is absent, even if the temperature is abnormally high and the non-paralysed parts are sweating profusely. Thus may arise hyperthermia or hypothermia. The former condition is much more common in patients placed in warm beds in a hospital; the latter condition is seen when the patient has been exposed to cold after the accident.

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# NEUROLOGICAL STUDIES.

(SECOND SERIES).

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EDITED BY

ARTHUR F. HERTZ, M.A., M.D. Oxon., F.R.C.P.,  
Assistant Physician, and Physician-in-Charge of the Neurological  
Department.

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1. CLINICAL VARIETIES OF DISSEMINATED SCLEROSIS: an Analysis of 50 Cases. By Arthur F. Hertz, M.D., and W. Johnson, M.D.
  2. THE QUANTITATIVE ESTIMATION OF THE VIBRATORY SENSE, and its Application in the diagnosis of Peripheral Neuritis. By J. L. M. Symms, B.A. Camb., M.R.C.S., L.R.C.P.
  3. MENTAL DEFICIENCY IN CHILDREN, with Special Reference to the Influence of Congenital Syphilis. By W. Johnson, M.D.
  4. CASE OF HEREDITARY INTENTION TREMOR. By Arthur F. Hertz, M.D.
  5. A CASE OF EPILEPSY WITH REMARKABLE AURA. By Arthur F. Hertz, M.D.
  6. A CASE OF LEAD NEURITIS INVOLVING THE CIRCUMPLEX NERVES. By Arthur F. Hertz, M.D.
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## 1. CLINICAL VARIETIES OF DISSEMINATED SCLEROSIS.

AN ANALYSIS OF 50 CASES.

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By

ARTHUR F. HERTZ, M.D., AND W. JOHNSON, M.D.

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THE well-known picture of fully-developed disseminated sclerosis with spastic paraplegia, intention tremor, nystagmus, scanning speech, primary optic atrophy and areas of anæsthesia is rarely present until the disease has existed for several years. At this

stage little or nothing can be done in the way of treatment. On the other hand, in the earlier stages considerable improvement may result and the rate of progress of the disease may be considerably diminished by suitable treatment, complete rest and the administration of arsenic being of most value. It is consequently of great importance to recognise the earliest signs of the disease. It is only in recent years that the extraordinary variety of the initial symptoms has become known and the fact become recognised that many cases formerly diagnosed as primary spastic paraplegia, primary ataxic paraplegia, primary optic atrophy, ophthalmoplegia and hysteria were really cases of disseminated sclerosis.

In an out-patient department fully-developed cases of disseminated sclerosis are rarely seen, as the patients are unable to get about with sufficient ease. On the other hand, numerous cases are seen in the early stages, as, with the exception of the syphilitic and parasyphilitic diseases, disseminated sclerosis is the most common organic disease of the central nervous system. Remembering this, it is interesting to recall the fact that the disease had never been recognised in England until Moxon described eight cases, with two post-mortems, in the Guy's Reports for 1875. In the present paper we have collected together all the cases seen in the Neurological Department since it opened in 1910, and we have added a few cases seen in private in order to bring the number up to fifty. After a brief discussion of ætiology and symptoms we have attempted to classify the cases according to the most prominent early symptoms, and have described in some detail one typical example of each variety. We hope at a later date to publish another series of 50 cases, in each of which every symptom has been investigated in greater detail.

#### ÆTIOLOGY.

1. *Age and Sex.*—Of our 50 cases, 19 occurred in males and 31 in females. The following table shows the distribution of

the cases according to the age at which the first symptom appeared :—

Age of onset	10-15	15-20	20-25	25-30	30-35	35-40	40-45	45-50	50-55	Total
Males ...	1	2	4	4	3	2	2	1	0	19
Females ...	2	5	3	10	4	3	1	1	2	31
Total ...	3	7	7	14	7	5	3	2	2	50

The average age of the 50 cases is 29·6, that for the males being 29·7, and for the females 29·5. The oldest male was 46 and the youngest 15 ; the oldest female was 56 and the youngest 14.

2. *Heredity*.—There was no evidence in any of our cases of any hereditary disposition to the disease.

3. *Infections*.—In two cases the first symptoms occurred immediately after an attack of influenza, and in a third influenza was the cause of a sudden return of symptoms after a considerable period of quiescence. The Wassermann reaction was positive in two out of twelve cases in which it was tried, and in another it was positive at the first examination, but negative a few weeks later. As the two positive cases were quite typical examples of disseminated sclerosis, and as they did not respond at all to treatment with mercury and iodides, it is extremely improbable that syphilis had anything to do with the nervous disorder. The absence of a history of syphilis and of all other evidence of that disease indeed makes it doubtful whether the positive reaction even proves that the patients had ever been infected.

4. *Psychical Causes*.—In three cases the disease began soon after a fright, in two cases after an accident and in one case after an operation for hæmorrhoids. In the latter three cases the mental effect rather than the injury or operation itself was probably the important factor. In an additional case fright was the cause of a return of symptoms after their temporary disappearance.



5. *Parturition and Pregnancy*.—In one case the first symptoms appeared after a confinement, in a second after premature labour, and in a third a rapid advance occurred during pregnancy.

6. *Intoxications*.—In no case was there any evidence that lead, alcohol or other poison, except the influenza toxin, had any influence on the disease.

7. *Fatigue and Chill*.—Fatigue appears to have aggravated the condition in several cases, as might be expected from the fact that rest generally leads to more or less improvement. Exposure to chill was not a causal factor in any case.

#### SYMPTOMS AND SIGNS.

1. *Tendon phenomena and reflexes*.—The *knee-jerks* were increased in all cases except one, in which they were at first exaggerated, but gradually became weaker until the jerk was completely lost on one side and was very feeble on the other. The *ankle-jerks* were present in all cases except in the one just mentioned; in this case the jerks were present at first, but both finally disappeared. *Ankle clonus* was noted in 20 cases.

The *plantar reflex* was extensor in 41 cases on both sides, and in two on one side, the other side being doubtful. In four the big toe did not move on either side, even after the feet had been kept for some time in hot water; in one of these the soles were completely anæsthetic and there was no reflex at all, not even the tensor vaginae femoris contracting. In only three cases was the plantar reflex definitely flexor.

The *abdominal reflex* was absent on both sides in 24 cases and on one side in one case; in 8 cases it was present. No note was made about it in 17 cases. It was lost, therefore, on one or both sides in 75 per cent. of cases. With the exception of the exaggerated knee-jerks and the extensor plantar reflex, the loss of the abdominal reflex is thus the most common sign of disseminated sclerosis. It disappears, moreover, at a very early stage.

2. *Nystagmus*.—Nystagmus was present in 32 cases. In several it was very slightly marked, and in only a very small proportion did it occur when the individual was looking straight forward. In some cases it was merely lateral, in others it was rotary, and in a few it occurred when the eyes looked upwards as well as to the side. In many cases it was much more marked in one direction than the other and in one eye than the other.

3. *Tremor*.—Tremor, which in nearly all cases was intentional in character, was present in 24 cases. In a few it was so slight that it could be recognised only by special tests, but as a rule the patient had experienced some difficulty in pouring fluids and in writing.

4. *Speech*.—In 17 cases the speech was abnormal. In only a small proportion of these, however, was it scanning; most frequently it was simply monotonous and slow.

5. *Vision*.—Vision was impaired in 16 cases. In only a small proportion of these was definite *optic atrophy* present, and in several instances the loss of sight was only temporary. *Diplopia* was noted in 13 cases; in the majority of these it was due to definite paralysis of the external rectus muscle of one side. In several instances there was no diplopia and no ocular paralysis when the patient was seen, but a history was obtained of temporary diplopia, which had been present some years before the onset of other symptoms. The pupils were only rarely affected; they were occasionally unequal and responded feebly to light, but the Argyll-Robertson pupil was never observed. In a number of cases the pupil, after contracting to light, immediately dilated again, but not to the full extent. We are not yet certain as to whether this sign has any special diagnostic value.

6. *Abnormalities in micturition*.—Abnormalities in micturition were present in thirteen cases. They were generally so slight that the patient only mentioned them after special inquiry had been made. The most common type of abnormality was hesitancy, the patient having difficulty in beginning micturition, and taking an abnormal time to complete it. True incontinence, though not uncommon in the late stages, was only present in one of our cases.

7. *Astereognosis*.—Astereognosis was present in a marked degree in eight cases. As a rule only one hand was affected, or the abnormality in the other hand was comparatively trifling. The patient was completely unable to recognise the nature of objects placed in the hand, the judgment of size, weight and consistence being lost. In our experience disseminated sclerosis is the most common cause of astereognosis. In a considerable number of other cases some other abnormality of sensation was present, but this was generally ill-defined and inconstant.

8. *Mental symptoms*.—A large proportion of the female patients and a much smaller proportion of the male patients were abnormally emotional, frequently laughing and crying without sufficient reason. There appears, in fact, to be a definite mental condition, which is highly suggestive of disseminated sclerosis, but it is apt to lead the unwary to the diagnosis of hysteria. A few of the patients were definitely hysterical; some had hysterical symptoms, which were cured by suggestion, added to the organic symptoms. Most of the patients are inclined to be optimistic and even to laugh at their symptoms.

9. *Vertigo*.—Giddiness is an exceedingly common symptom and does not seem to bear any definite relationship either to ataxy or nystagmus.

10. *Other symptoms*.—In one case *impotence* was complained of; this symptom is probably not uncommon, but was not inquired into in the majority of our cases. In a few cases the patients had had *syncopal attacks*, and in one had had *epileptiform convulsions*. In one case *glycosuria* was present; this may, perhaps, have been due to a patch of disseminated sclerosis in the medulla. One patient suffered from typical *Raynaud's disease*, its onset having occurred at the same time as that of the other symptoms.

#### CLINICAL VARIETIES OF DISSEMINATED SCLEROSIS.

It is exceedingly difficult to classify all cases of disseminated sclerosis into the different types, as they all tend sooner or later to develop the classical symptoms, first described by Charcot, of

the well-marked cerebro-spinal form. In the early stages, however, many of the cases present themselves under such exceedingly different forms that a knowledge of the different types is of great value as an aid to the early recognition of the disease. The following classification cannot be considered as final, as there are probably other types which do not happen to have been represented in our fifty cases.

1. *Cerebro-spinal Type*.—The chronic cerebro-spinal type is the most widely recognised, although not the most common. In it the classical symptoms of spastic paraplegia, intention tremor, scanning speech, and nystagmus are present when the patient first comes under observation. It is so familiar that it is unnecessary to describe a case here.

2. *Spinal Type*.—(a) *Dorsal*. i. *Spastic*.—Sixteen cases, ten males and six females, presented a picture of spastic paraplegia when first seen. Some of these should probably be included in the cerebro-spinal group, but in a number of them other signs and symptoms were at first absent, or so slightly marked that they would have escaped observation if they had not been specially looked for. It is a good rule always to think of disseminated sclerosis when a patient between the ages of 15 and 45 has symptoms and signs of spastic paraplegia in the absence of any evidence of disease of the spine or spinal meninges. Indeed a diagnosis of primary spastic paraplegia should never be made, as a large proportion of cases, in which this diagnosis would formerly have been made, finally prove to be disseminated sclerosis.

William T., labourer, aged 39, has complained for seven months of gradually increasing stiffness of his legs, and for six months has on account of this been unable to work. His arms are strong, but he is too slow on his legs. He occasionally complains of a sensation of pins and needles down his legs. He has recently staggered in the dark, so that now he never goes out at night. Quite recently he has, to use his own expression, "staggered a bit in his speech," but it is not obviously abnormal to anybody who does not know him. For a short period his urine dribbled away during the day, but not at night; this symptom has now disappeared. There is no intention tremor, but there is a very slight loss of muscle-sense in the left arm and distinct exaggeration of the

arm-jerks on both sides. The legs are definitely spastic and the knee-jerks greatly exaggerated, and he has well-marked ankle-clonus. The plantar reflex is extensor, and the abdominal reflexes are absent. Vision is normal; the retinae, pupils and ocular muscles are normal, but there is a very slight degree of nystagmus when the patient looks to the right.

ii. *Ataxic paraplegic*.—In one man and two women the disease presented itself under the aspect of ataxic paraplegia, the gait of the patients showing the characteristic combination of ataxy with spasticity. In one of these cases the posterior column sclerosis progressed so much whilst the case was under observation that the knee-jerks and ankle-jerks, which were at first exaggerated, gradually diminished and finally disappeared, except for one knee-jerk, which could still be obtained with difficulty when the patient was last seen; the other symptoms showed that this was not a case of subacute combined degeneration of the cord. It is very doubtful whether such a disease as chronic primary ataxic paraplegia really exists, most cases formerly regarded as examples of that condition being really examples of disseminated sclerosis.

Alice O., aged 35, complained for two years of increasing difficulty in walking; during the last three months her eyesight has become less good than it was formerly, and she has become somewhat emotional. Her gait shows a combination of well-marked spasticity and ataxy. Her knee-jerks are increased and the plantar reflex is extensor, but there is no ankle-clonus. The abdominal reflexes are absent. There is no nystagmus, but there is slight intention tremor and a somewhat staccato speech, of which the patient herself was not aware, and scattered areas of diminished sensation are present in the legs and forearms. All the symptoms are, however, insignificant compared with the spastic ataxic gait.

(b) *Cervical*. i. *Ataxic*.—In six females tremor of one or both hands was the most marked or only obvious symptom, but in all the cases the diagnosis was confirmed by the presence of some other symptom or sign, which had often escaped recognition by the patient herself.

Mrs. B., aged 39, noticed about a year ago a slight shakiness of the right hand when she poured out tea. This gradually increased so that she has had to give up knitting, but she is still able to sew and to write, her writing being larger than it was and shaky, but quite easily legible. The tremor is absent when the arm is at rest, developing as soon as she tries to do anything. It is worse when she is tired, but not increased by

excitement. It cannot be controlled by the will. The last few weeks she has noticed a very slight tremor of her left hand. She complains of no other symptoms. Her eyes are normal in every way, and there is no nystagmus. The abdominal reflexes are absent. The knee-jerks and ankle-jerks are very exaggerated, but there is no clonus. There is a definite loss of sensibility for all forms of stimulation in the tips of the fingers and in the soles of the feet, a slight degree of astereognosis being present in the right hand. Even after the feet had been warmed the big toe did not move on stroking the soles of the feet; on the right side there was no reflex present at all, but on the left side there was a feeble contraction of the tensor vaginæ femoris.

ii. *Sensory*.—In four females astereognosis was the most prominent and for a time the only symptom. Though the diagnosis was suspected from the first, in two cases it was only cleared up when, after two years had elapsed, other signs and symptoms developed.

Rose A., aged 22, woke up one day in March, 1910, a week after having gone into service, with a numb and tingling sensation in her right hand. Sensation rapidly became so impaired that she could not feel articles in her hand, and consequently allowed them to drop. When examined in January, 1911, complete astereognosis was present in the right hand, the patient being quite unable to recognise objects placed in it. The patient complained of no other symptoms, except that she had to pass urine about every hour during the day. There was, however, well-marked rotatory nystagmus of both eyes, especially on looking to the right. The knee-jerks were normal and ankle-clonus absent; the abdominal reflexes were present and the plantar reflex flexor. There was no tremor, and speech and vision were normal. The possibility of a tumour in the cerebello-pontine angle was considered; the Wassermann reaction was found to be positive, although there was no other evidence of syphilis. In April the only change was loss of sensation in the right foot, which caused some unsteadiness in walking; the left side of the face was weak, and the tongue was tremulous. The Wassermann reaction was now negative. In August, 1912, there was still complete astereognosis of the right hand, but sensation to touch and pain were only slightly marked. All arm-jerks were greatly exaggerated, especially on the right side. The knee-jerks had become more brisk, but the plantar reflex was still flexor. The speech was now definitely scanning in character. Nystagmus was still well marked, and the left external rectus muscle was weak. The discs were normal, but peripheral vision was temporarily impaired in the left eye at the end of 1911.

3. *Cerebral Type*.—(a) *Hysterical*.—Several patients had been regarded at an earlier stage as being hysterical, and in a few it was extremely difficult when they were first seen to diagnose between hysteria and disseminated sclerosis.

Mrs. D., aged 41, had an operation for hæmorrhoids performed a year ago. Ever since then she has complained of a multitude of symptoms. She is extremely nervous and excitable, cries on being spoken to, and has neuralgic pains all over her; she feels as if her legs were giving way and complains of palpitation and flatulence. On examination the only evidence of organic disease was a very brisk knee-jerk on both sides, well-marked ankle clonus on the right and slight on the left side, extensor plantar reflex and absent abdominal reflex on both sides. The speech was normal, and nystagmus, tremor and eye changes were absent. The patient underwent a rest cure in December, 1910. She has been perfectly well since then, but the physical signs indicating organic disease of the nervous system are unaltered, and in all probability symptoms of disseminated sclerosis will develop at some future period.

(b) *Cerebellar*.—In some cases the symptoms are at first very suggestive of a cerebellar tumour.

Ethel P., aged 30, has been giddy for a year. Some months ago she had temporary difficulty in speaking and swallowing. Her arms and legs are quite strong, but her gait is staggering and highly suggestive of cerebellar disease. She has slight nystagmus and occasional diplopia. There is slight intention tremor and loss of muscle-sense. Although there is no weakness or spasticity in the legs, the knee-jerks and ankle-jerks are exaggerated, the plantar reflex is extensor and the abdominal reflexes absent.

(c) *Ponto-bulbar*.—The ponto-bulbar form of disseminated sclerosis is rare. The following case closely resembles one published by Oppenheim, who first recognised this type of this disease:—

Mrs. F., aged 50, has become very nervous and depressed during the last year. Three months ago the left side of her face became weak and numb. She saw double and there was left-sided ptosis. Soon afterwards she had some difficulty in walking, and both legs felt numb and occasionally gave way. The left arm has recently also become weak. Although her relations have not noticed any alteration in her speech, it is definitely staccato in character. On examination she was found to have left-sided paresis of the face, arm and leg. The left pupil is larger than the right, and nystagmus is present when the eyes are turned to the left. The tongue is tremulous. The muscles of mastication are normal, but there is a considerable degree of anæsthesia of both the skin and mucous membranes supplied by the left trigeminal nerve. There is some loss of sensation of the left arm, and both arms are slightly weak. The patient is giddy when she moves, and her gait is reeling; she is unsteady on standing. There is no intention tremor. The abdominal reflexes are absent. The left knee-jerk is very feeble and the left ankle-jerk is absent, but the jerks on the right side are normal; three months ago the knee-jerks were equal and exaggerated.

The plantar reflex on the left side is extensor; it could not be obtained on the right side. When seen again after resting for a month her power of walking had greatly improved and the tremor of the tongue and diplopia had disappeared; she now felt as if a film was over her eyes, although the discs are quite normal. The marked improvement in many of the symptoms appears to exclude a tumour in the ponto-cerebellar angle, which had at first been thought of as an alternative diagnosis.

4. *Optic Type.*—In four males and one female impaired vision was the first and most prominent symptom. In some cases the presence of disseminated sclerosis was only suggested by the presence of an extensor plantar reflex and the absence of the abdominal reflexes, no symptom other than the loss of vision being present. In all cases of loss of sight, whether associated with primary optic atrophy or not, the possibility of disseminated sclerosis should be considered, if no other obvious cause is present, particularly if the loss of vision spontaneously undergoes rapid and considerable variations in degree.

Agnes M., aged 41, complains of dimness of vision and feeling of oppression in the head, which have been slowly developing during the past year. These were her only symptoms, and she consequently went first to ophthalmic out-patients. It was found in August, 1910, when she was transferred to the Neurological Department, that in addition to double optic atrophy, the only symptom was well-marked nystagmus, which was present when the patient looked to either side and might easily have been a result of the defective vision. The arm-jerks and knee-jerks were all exaggerated; there was no ankle-clonus, but the plantar reflex was extensor on both sides. The abdominal reflexes were absent. When seen again in August, 1912, the nystagmus was constantly present whether the patient looked forward, upwards or sideways; it was always lateral in direction and only ceased when the eyelids closed. The patient had recently developed a slight intention tremor, so that she was liable to drop objects and spill fluids; her gait was slightly ataxic, and she had occasional frequency of micturition. Her speech was still normal.



## 2. THE QUANTITATIVE ESTIMATION OF THE VIBRATORY SENSE AND ITS APPLICATION IN THE DIAGNOSIS OF PERIPHERAL NEURITIS.

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By

J. L. M. SYMNS, B.A. (Cantab.), M.R.C.S., L.R.C.P.

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WHEN one considers the diagnosis of lesion of the sensory tracts of the spinal cord, one is struck by the fact that the ordinary clinical methods of investigation can show no conditions in which the fibres are abnormal, but still carrying on their function. There is, however, one tract which lends itself to more accurate investigation. Egger, in 1899, called attention to the sense of vibration felt when the foot of a vibrating tuning fork is placed over subcutaneous bony points.

The investigations of Bing show that the sensation is probably conducted upwards in the posterior columns of the white matter by fibres that do not decussate. In his first case there was an extradural unilateral fibrosarcoma of the cervical region of the cord present. There was diminution of tactile sensation on the side opposite to the lesion, and loss of the sensation of pain and temperature. On this side the vibratory sense was present. On the same side as the lesion there was loss of the vibratory sense, but the sense of pain and temperature were normal.

Bing's second case was one of traumatic hæmatomyelia, in which the vibratory sensation was normal, but there was marked

impairment of the sense of pain and temperature. The post-mortem showed that the grey matter of the cord was entirely destroyed at the site of the lesion.

In this country Williamson has shown that the vibratory sensation is often lost when other forms of sensation are present. He states: "The vibratory sensation is a delicate test for detecting slight impairments of sensation. The vibratory sensation may be lost when other forms of sensation are felt quite well or are only slightly impaired. This is sometimes the case in early tabes, in slight peripheral neuritis, and often in diabetes mellitus."

The ordinary method of investigating the vibratory sensation does not show the abnormal states which precede the total impairment of the tract. However, with this sensation there are not the technical difficulties that are met with in the investigation of the thermal or other sensations.

In my investigations I have used the tuning fork introduced by Edelmann, of Munich, for use in Schwabach's aural test for the absolute bone conduction of hearing. Its vibration rate is 108.75 per second. Two pieces of steel are attached to the upper portion of the fork in such a manner that when the arms of the fork are vibrating a small window is seen between them. This disappears when the vibrations of the fork reach a definite amplitude. The moment that the window disappears I apply the fork to the subcutaneous bony point, and start a stop watch. The patient is instructed to give a signal at the moment when he ceases to appreciate the vibration; the watch is then stopped and the time recorded. The subcutaneous bony points taken were those recommended by Williamson, viz., the internal malleolus, external malleolus, the tibia, the anterior superior spine of the ilium, the bottom of the sternum, the radius, and the ulna.

By this method on thirty normal individuals I found what period elapses before the sensation disappears at each bony point.

The average periods, together with the maximum and minimum, are shown in the following table:—

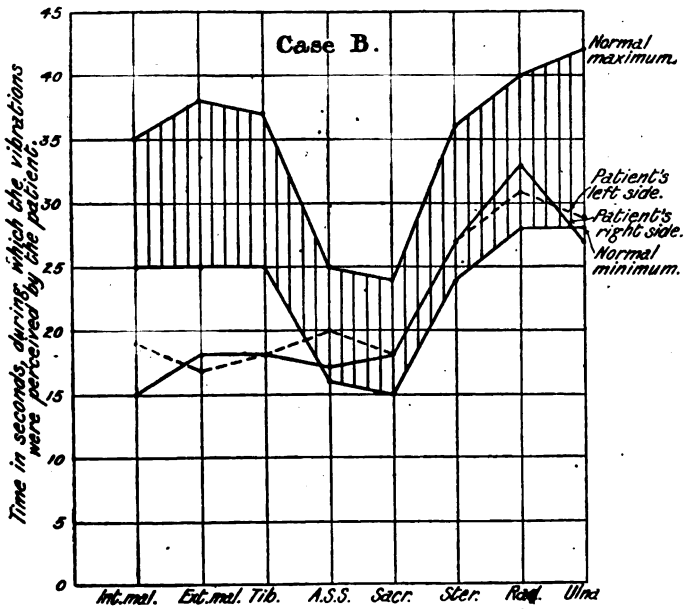
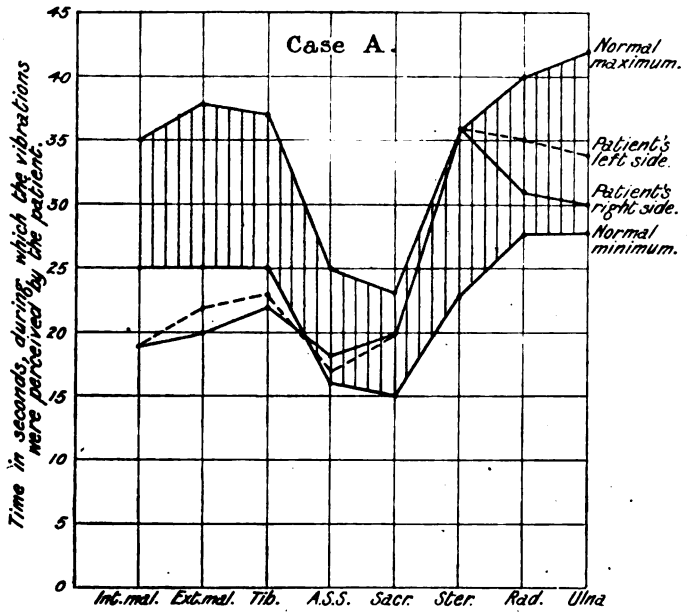
	Average.	Maximum.	Minimum.
Internal malleolus .. ...	27	35	25
External malleolus ... ..	28	38	25
Tibia (middle of shaft, inner aspect) ... ..	29	37	25
Anterior superior spine of ilium ... ..	21	25	16
Sacrum ... ..	20	23	15
Sternum .. ...	30	36	24
Radius (lower end) ... ..	33	40	28
Ulna (lower end) ... ..	34	42	28

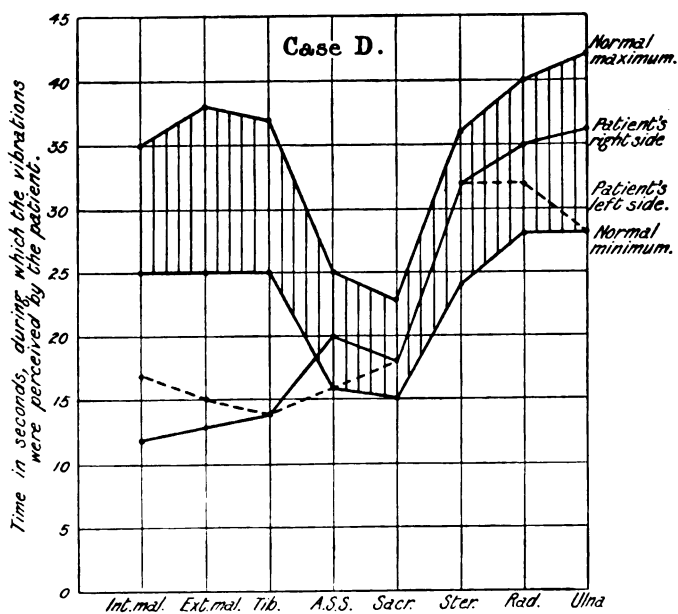
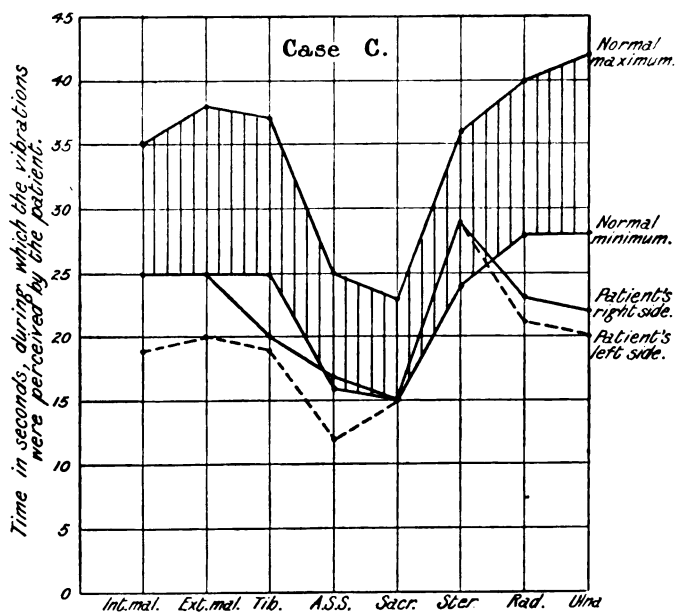
When this test is applied to patients suffering from impairment of the vibratory sensation, a numerical ratio to the average normal is obtained which represents the degree of impairment. In the application of this test to patients suffering from lesions causing impairment of their vibratory sense I have used a graphic method. The following are some illustrative cases. The height in the accompanying curves represents the time during which the sensation was appreciated. Superimposed is the normal maximum and minimum for comparison.

Case A.—A man, aged 34, who complained of a sensation of tingling in his hands and forearms. This patient's knee-jerks were normal, and there was no muscle tenderness; he confessed to being an excessive drinker. On testing his vibratory sense there was considerable diminution over both legs from the knee downwards.

Case B.—A man, aged 34, who worked as a painter. He complained of constipation. There was a slight blue line on his gum. The jerks were normal. On testing his vibratory sense there was considerable diminution over both lower limbs.

Case C.—The man, aged 32, suffered from right-sided wrist drop. He had a definite blue line, and was very constipated. His jerks were normal. His vibratory sense showed diminution in both arms. The legs were also affected, the right to a less extent than the left.





Case D.—A man, aged 53, suffering from diabetes mellitus. He was passing 21.7 grms. of sugar per diem on a diet containing 62 grms. of carbohydrate. He complained of pain following the course of his right sciatic nerve, but there were no other indications of peripheral neuritis. His vibratory sense was impaired over both lower limbs, slightly more on the right than on the left.

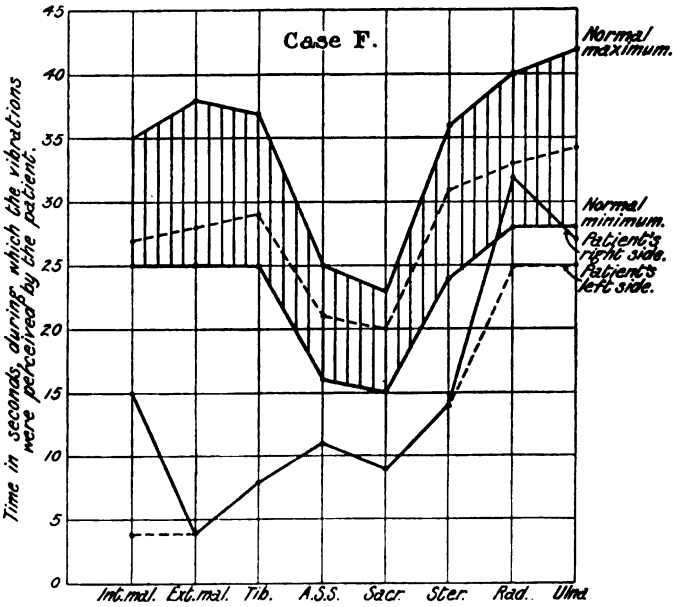
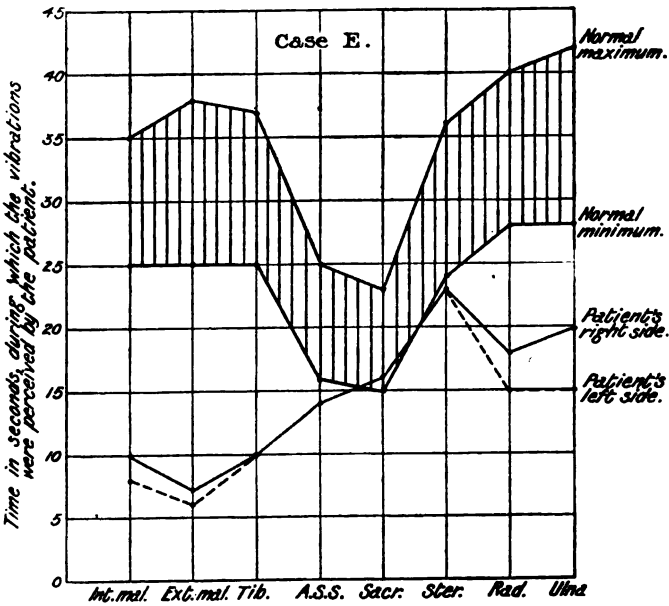
Case E.—A man, aged 37, who complained of depression and tinglings in his arms and legs. His knee-jerks and tendo-Achillis-jerks were absent. He acknowledged being a large drinker. There was some tenderness of the calf muscles. His vibratory sense was very much decreased over both lower limbs, and to a less extent over his arms.

Case F.—A woman, aged 60, who complained of joint pains, vomiting, and headaches. There was marked tenderness in her calf: knee-jerks and tendo-Achillis-jerks were absent. The vibratory sense was decreased throughout except in the right arm. The sacrum, sternum, and anterior superior spines were involved.

There are points in the preceding curves which are typical of peripheral neuritis. The vibratory sense is impaired over the extremities first, and in a disease which causes general peripheral neuritis impairment is first noted in the lower extremity. The sacrum is not involved until there is marked general involvement of the vibratory sense, and the diagnosis is obvious.

This impairment of the vibratory sense over the sacrum I have always found in *tabes dorsalis*, and it has aided in the differential diagnosis of those cases of peripheral neuritis simulating *tabes*.

I have seen several cases in which this test has shown early peripheral neuritis involving the sciatic nerve in patients who considered they had injured their leg during their employment.



CONCLUSIONS.

1. Decrease in the vibratory sense shows that the disease is involving sensory path.
  2. A decrease in the vibratory sense is usually found in cases in which peripheral neuritis is a complication.
  3. The vibratory sense is only affected over the sacrum at a stage when the jerks are absent and the diagnosis is certain.
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### 3. MENTAL DEFICIENCY IN CHILDREN, WITH SPECIAL REFERENCE TO THE INFLUENCE OF CONGENITAL SYPHILIS.

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By

W. JOHNSON, M.D.

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THE term "mental deficiency" as applied to the following collection of cases does not include the well-marked cases of idiocy and imbecility which we are accustomed to see in asylums. It is here confined to those children who are described as "backward" by their parents and who, at school, have been unable to keep pace with a class consisting of children of their own age. They are accordingly sent to the special schools, such as exist in London, or failing this refuge they are kept at home, where, being amidst unfavourable surroundings, their condition lapses from bad to worse. During the past eighteen months some twenty-five such children have come under investigation in the Neurological Out-Patient Department.

The ætiology is admittedly difficult and complicated. One well-defined group of cases is that in which the mental impairment is secondary to some organic disease. Amongst our cases the most prolific cause in this group was epilepsy. Other causes were spastic diplegia, infantile hemiplegia—in one case associated with signs of congenital syphilis—Mongolism, cretinism, and complete nerve deafness. These conditions accounted for just over half of the total cases collected. They are not considered further here. The remainder, eleven in number, showed no sign of organic disease. Physically they were and always had

been fairly healthy children. In three cases only was a history given of convulsions having occurred in infancy. In two there was some evidence of congenital syphilis in the form of notched incisors, but none of the other stigmata were present. In no case had the mental impairment directly followed a serious illness, although in one or two instances such an illness had apparently exaggerated the mental condition.

The history usually obtained was that the child was apparently healthy at birth and thrived well. Walking and talking were both slowly acquired, as a rule, not before eighteen months to two years. One child made no effort to talk before five years of age. Other minor abnormalities, such as attacks of temper and screaming fits on the slightest provocation, transient strabismus, and often incontinence of urine lasting till the child was three or four years old, had been noticed during infancy. In three cases only had a vacant expression in the eyes been remarked during early life. In most of the cases it was with the commencement of school that the relatives realised the fact that the child was deficient. Inability to acquire the arts of reading and writing was present in all cases. Some could only copy letters, others write their names in a very shaky hand, and this only after infinite pains had been expended on teaching them. So at the age of five they were usually stamped as being definitely deficient through their inability to learn. Later years brought forward further evidence in the form of a disordered state of the mind. Certain cases were fond of solitude, would sit about alone, and, if disturbed, would refuse to be drawn into conversation. They behaved spitefully to their brothers and sisters, and were subject to attacks of sulks or temper. Sometimes these attacks were destructive in nature, and it was necessary to keep continual watch over them.

With strangers their deficiency appears to be more marked than it really is. They easily get confused, and their answers rarely get beyond a monosyllable, and this is often whispered. They hesitate over giving their own name, and very few could

give the day in the week. Their whole condition is "hopelessly backward."

The following notes are taken from typical cases:—

1. Rose W., aged 17, was a well-developed and bright infant, no instruments having been used at birth. She neither walked nor talked before 18 months. Slight strabismus developed when two years of age, and has continued up to now. She has never had any fits. Progress at school was very slow, and she was sent to a special school. She is a tall child, and her head is inclined to be large. The palate is slightly arched. Teeth are normal. Ophthalmoscopic examination of the eyes showed nothing abnormal. She is quiet and reserved and sits much alone. Her mental state is simply one of childishness. She is no trouble to manage and seems perfectly contented. The mother says she has not improved at all in intelligence since she was ten years of age.

2. Chas. S., aged 11, was well developed at birth, at which instruments were used. He has never had any fits. He began to talk when 2 years old. He is quite unable to read or write, and cannot answer the simplest questions. His memory is very poor. He is mischievous and destructive, and requires constant supervision. He is small for his age. There is excess of hair about the face. His teeth are normal. The eyes also show nothing abnormal.

Inquiry into the family history in several cases suggested the presence of a syphilitic taint, and accordingly a Wassermann test on the blood-serum was performed in nine out of the eleven patients. It was positive in each. Further tests were then done on relatives of the patients with the following results:—

1. Rose W., aged 17. The mother has had one miscarriage and gives a positive Wassermann reaction. There are two other children, one a boy of 15, who has epilepsy and is decidedly deficient mentally, and the other, a boy aged 12, who is bright and intelligent and doing well at school. In both of these children the Wassermann reaction was positive.

2. Chas. S., aged 11. The mother has had one miscarriage; the Wassermann reaction of her blood was negative. There is one other boy, 14 years of age, who is a perfectly normal child. His blood gave a positive Wassermann reaction.

3. Violet S., aged 14. The mother had nine children, four of whom died in infancy, and no miscarriages. One sister, the only one who could be prevailed upon to be tested, gave a positive Wassermann reaction. She is quite a bright child.

Of the six remaining cases, the mother of the first gave a positive reaction, the brother of the second a positive reaction, while in the third the family history showed that the remainder

of the family—three brothers and sisters—had died in infancy. No relatives of the last three cases would come forward to be tested.

To draw hard and fast conclusions from these results would be injudicious at the present moment, when the value of the Wassermann test is being disputed by certain high authorities. But I must here acknowledge my indebtedness to Dr. Gordon Goodhart, who, with one or two exceptions, has performed all the tests himself, thus reducing the personal error in the results to a minimum. Moreover, he makes use of the original Wassermann method, which for diagnostic purposes is admitted to be more reliable than any of the modified methods.

The type of syphilitic taint in these cases is necessarily of the parasyphilitic variety, in which we have first an arrest in development and later a definite decay in those cerebral neurones which are responsible for memory and the intellectual functions. It will be interesting to observe whether any of these patients ever progress far enough to become juvenile general paralytics. In only one of the cases was there ever any suspicion of this disease. The pupils reacted sluggishly to light, but no more definite sign of the disease has yet developed.

In keeping with the above results is the fact that the Wassermann reaction is more often positive in parasyphilis, and especially general paralysis, than in acquired syphilis.

As regards the other assigned causes of mental degeneracy, such as alcoholism, phthisis, epilepsy, and neurasthenia in the parents, it is difficult to draw conclusions, as few families are to be found who do not give a history of one or other of these conditions. In any case, syphilis would seem to bear a major share in the ætiology. Adenoids alone cannot be regarded as a cause of advanced mental deficiency, as the slightly arched palate is too common a condition in an out-patient department, but it may be contributory.

So far as I have been able to judge the effect of treatment in my cases, there is not much to be hoped for. We have given mer-

cury in the form of liquor hydrargyri perchloridi a half to one drachm three times a day over extended periods, even up to six months. In some cases no improvement was noted; in others the general health certainly improved, and the child became more cheerful. I have one letter from a schoolmaster in which he states the boy definitely improved in intelligence and ability. Otherwise, all that can be done is to educate the patient to follow some manual employment, preferably out of doors. In this capacity lies his best outlook, but his lack of attention and inability to apply himself for continuous effort must always greatly handicap him.

#### 4. CASE OF HEREDITARY INTENTION TREMOR.

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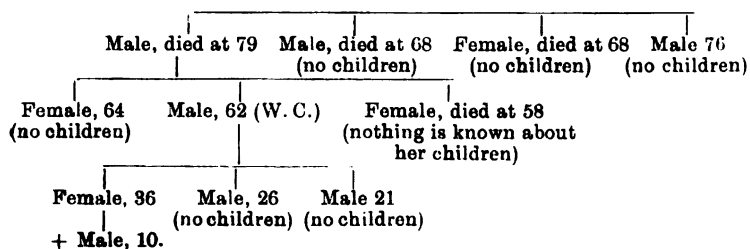
By

ARTHUR F. HERTZ, M.D.

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W. C., an engine-fitter, aged 62, has suffered from a tremor of his hands since the age of four and possibly earlier still. During the last five or six years it has been getting worse. A very slight tremor is occasionally present in the legs and head, and the voice has a somewhat tremulous character, but there is no tremor of the face or tongue. Apart from the tremor the patient is perfectly healthy, and there are no other signs of nervous disease. The tremor is absent when the hands are resting upon the knees; as soon as they are held out the tremor begins. It is at first fine, but becomes coarser as the arms get tired. It is increased when voluntary movements are performed, being strikingly similar in character to the "intention tremor" of disseminated sclerosis. It is aggravated by excitement and cannot be controlled by the will. As the accompanying family tree shows, the patient's father, his father's only two brothers and only sister, both of his own sisters, both of his sons and his only daughter suffer or suffered from the same condition. He had no brothers, and his paternal uncles and aunts had no children. One of his sisters had no children, and he knows nothing about the children of his other sister. His daughter, who is aged 36, is the only one of his children who is married; her only child, a boy aged 10, has no tremor. In the case of

his own three children the tremor started in earliest infancy, and he believes that it started in childhood in the other members of his family.



+ indicates the only member of the family with no tremor.

## 5. A CASE OF EPILEPSY WITH REMARKABLE AURA.

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By

ARTHUR F. HERTZ, M.D.

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ESTHER D., aged 31, has had typical major epileptic fits since she was 14 years old. During the first year she had only two, but the frequency gradually increased, until during the last five years she has had four or five a fortnight alternating with free intervals of a fortnight. The fits are preceded by a remarkably prolonged aura. This begins with a feeling of pins and needles in the second finger of the right hand, which gradually passes up the right arm, reaching the front of the elbow in twelve hours and the axilla in twenty-four hours. In another twelve hours it reaches the upper end of the sternum, where a "catchy feeling" in the throat, which the patient describes as resembling a "hiccough without the noise," is felt two or three times an hour. In another twelve hours the left axilla is reached, then the left elbow and finally, about three days after the onset of the aura, the tip of the second finger of the left hand; as soon as this point is reached the fit occurs. The sensation of pins and needles lasts about a day in each situation and is followed by a sensation of numbness of equal duration. The catchy feeling in the throat continues to recur until the fit occurs.

The patient was instructed to tie a string tightly round the base of the second finger of the right hand as soon as the tingling sensation was felt at its tip, and, if this did not stop its progress, she was told to tie the ligature successively round the elbow and axilla of the right side and the axilla, elbow and finger of the left side in front of the advancing aura. No bromide was given. The patient was able to stop the aura on every occasion by tying the ligature round the base of the second finger of the right hand, the sensation of pins and needles disappearing in about twelve hours.



## 6. A CASE OF LEAD NEURITIS INVOLVING THE CIRCUMFLEX NERVES.

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By

ARTHUR F. HERTZ, M.D.

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A HOUSE painter, 60 years old, complained of inability to raise his right arm, which had developed gradually in the last few months. The right deltoid was very weak and considerably atrophied. The left deltoid was also weaker than normal, although the patient did not spontaneously complain of this. No other muscles were affected, and the extensors of the wrists and fingers were quite normal. The arm-jerks were unobtainable, the knee-jerks normal and the ankle-jerks absent. A well-marked blue line was present, and the hæmoglobin percentage was 65. The fact that the left deltoid was slightly affected as well as the right and the absence of the ankle-jerks showed that the paralysis of the right deltoid was due to neuritis, the circumflex nerves being principally involved. The occupation of the patient and the blue line on his gums indicated that the neuritis was due to lead poisoning.

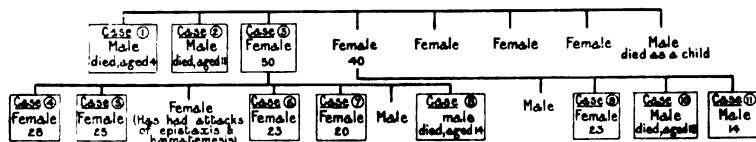
# HEREDITARY FAMILIAL CONGENITAL HÆMORRHAGIC NEPHRITIS.

By

GEORGE KENDALL, L.R.C.P., M.R.C.S.,

AND

ARTHUR F. HERTZ, M.A., M.D., F.R.C.P.



THE above tree is of a family, three out of eight members of which in one generation and eight out of eleven in the next generation suffered from nephritis. Those enclosed within a square were affected, whilst the others, so far as we are aware, were unaffected. For the details of Cases 3, 5, and 8 we are indebted to a paper by Dr. Leonard G. Guthrie, read before the Harveian Society of London on April 24th, 1902,<sup>1</sup> and he has kindly furnished us with further details about Case 8. Case 10 was under the care of one of us (G. K.) during his last illness, and Case 11 has been seen by both of us. The incomplete notes regarding the other cases are derived from what the mother of Cases 9, 10, and 11 has told us.

Case 1.—A male, the eldest of the first generation, died when four years old, after having had albumen and blood in his urine. The cause of death is not known.

Case 2.—A male, died when eleven years old, after having had albumen and blood in his urine. The cause of death is not known.

Case 3.—Female, 50 years old, the mother of Cases 4, 5, 6, 7, and 8, said, in 1902, that as long as she remembered she had been subject to attacks of hæmaturia, similar to those of her children. They were always produced by eating black currants and drinking claret and also accompanied various slight ailments. The last occasion on which severe hæmaturia occurred was in 1896, shortly after the birth of her youngest child. She always appears to be well, and is active and energetic.

Case 4.—Female, 28 years old, is subject to attacks of hæmaturia.

Case 5.—Female, 25 years old, has had albuminuria at least since the age of 12; the urine has also always contained traces of blood detected on microscopical examination, fragments of blood casts, but no hyaline or granular casts. When 13 years old, a week after she had had for a few days a severe sore throat, she was violently sick, her tongue was dry, and her temperature rose to 102° F.; she complained of headache, and passed bright scarlet urine. The temperature gradually fell, and the urine regained its normal appearance in about a fortnight. There was never any cedema, the heart was normal in every way, and the kidneys were not palpable. Black currants are said to have induced similar attacks of hæmaturia.

Case 6.—Female, 23 years old, is subject to attacks of hæmaturia, which can always be brought on by the consumption of black currants.

Case 7.—Female, 20 years old, is subject to attacks of hæmaturia, which can always be brought on by the consumption of black currants.

Case 8.—Male died in 1910, aged 14 years, from acute pericarditis. His urine was never completely free from albumen and blood. It was always red and never smoky in appearance. Blood casts, but no hyaline casts were present. At any rate up to the age of twelve there were no cardio-vascular abnormalities. Every two or three months an attack lasting about a week occurred, in which the amount of blood in the urine was greatly increased; slight malaise was present, and the tongue was furred. These exacerbations were accompanied by enuresis and frequency of micturition, and were often brought on by catching cold. Attacks also followed influenza, extremes of hot and cold weather, the consumption of strawberries and asparagus, but not black currants, gooseberries, raspberries, or grapes. Even when severe hæmaturia was present he was as "bright and merry as possible."

Case 9.—Female, 23 years old, has had albumen in her urine since she was three weeks old. There has never been any obvious hæmaturia, and her general health appears to be excellent.

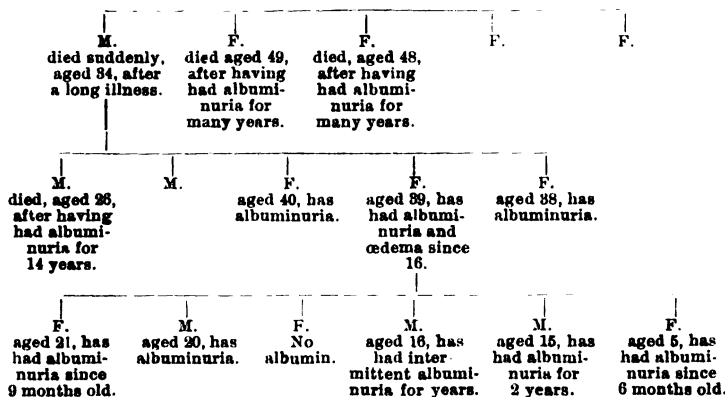
Case 10.—Male died in 1911, aged 19. He never had scarlet fever, and was accidentally discovered to have albumen in his urine after a football match when 14 years old. The albuminuria persisted, and attacks of hæmaturia frequently occurred after violent exercise, but he appeared to be otherwise in excellent health and was a first-class cricketer and footballer. At Christmas, 1911, he had a bad attack of influenza; he played in a badminton tournament before he had completely recovered and developed uræmia, from which he died.

Case 11.—Male, 14 years old, was discovered when two years old to have albuminuria; since then albumen has been found whenever the urine has been examined. In spite of this he is full of life and spirits and plays games as well as any average boy. There has never been any œdema. The kidneys are not palpable, and the heart is normal, but the blood pressure is 143 mm. of mercury. The urine is always clear and there has never been blood visible to the naked eye. Its specific gravity

is 1010; 0·4 per cent. of albumen is present, and this is about the same quantity as four years ago. Only a small deposit is produced on centrifugalisation; it contains numerous red blood corpuscles, the proportionate number of leucocytes, a small number of granular and hyaline casts, some excess of mucus and the normal urinary epithelial cells, but no renal epithelium, pus or other abnormality.

We have called this condition "hereditary, familial, congenital, hæmorrhagic nephritis": hereditary, because at least two generations have been affected; familial, because it has attacked several members of the same family; congenital, because abnormal urine was discovered in one case when the patient was only three weeks old, in another when the patient was two years old, and in the remaining cases in early childhood and apparently on the first occasion that the urine was ever examined. "Hæmorrhagic nephritis" is, perhaps, open to criticism, but we think that the name corresponds better with the facts than "Congenital, hereditary, and family hæmaturia," under which Dr. Guthrie published his cases, for in Case 11 there has never been obvious hæmaturia, only traces of blood, insignificant when compared with the amount of albumen having been found on microscopic examination. In several of the other cases, moreover, albumen has been present without obvious blood in the intervals between the attacks of hæmaturia. Case 10 died from uræmia and Case 8 died of acute pericarditis, which may quite conceivably have been associated with nephritis. Unfortunately in neither case was a post-mortem examination made. In Case 11 the unusually high blood pressure in a boy of 14 points to the presence of definite nephritis, which is confirmed by the presence of hyaline and granular casts. It seems justifiable to call the nephritis hæmorrhagic, as most of the cases have been characterised by recurrent attacks of hæmaturia, the amount of blood passed in some cases being so great that the urine resembled that of a patient with renal calculus or a growth rather than nephritis.

We have only been able to find records of two other families affected in a similar manner. The following tree is that of a family, an account of which was published by Dickinson<sup>2</sup> in 1875.



Attlee<sup>3</sup> in 1901 described the history of three sisters, aged 5, 4, and 2, each of whom passed urine, which constantly contained albumen, often faint traces of blood, and occasionally granular casts; they suffered from intermittent attacks of hæmaturia, which were associated with malaise, and sometimes with vomiting and slight pyrexia. None of them showed any cardio-vascular changes. Their father had died at the age of 30 from uræmia.

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2. W. H. Dickinson, *Diseases of the Kidney*, I., 379, 1875.
3. W. H. W. Attlee, *St. Bartholomew's Hospital Journal*, ix., 41, 1901.



# CONGENITAL INTESTINAL OCCLUSION.

AN ACCOUNT OF TWENTY-FOUR UNPUBLISHED  
CASES, WITH REMARKS BASED THEREON AND  
UPON THE LITERATURE OF THE SUBJECT.

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By

N. I. SPRIGGS, M.D., B.S. (Lond.), F.R.C.S. (Eng.).

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## PREFACE.

THE subject of this paper was suggested to the writer by his seeing two cases of congenital intestinal occlusion at Guy's Hospital a few years ago. The other cases have been collected from the museums of the Royal College of Surgeons and of some of the London hospitals. A few of the cases have been previously published by the writer in a shorter paper. Four cases which, strictly speaking, should not be inserted in the series (Cases 1a, 1b, 8a, and 14a) are appended because of their especial interest, but they are not counted in making up the twenty-four. Pyloric and ano-rectal occlusions are excluded from consideration.

This paper does not claim to be exhaustive. Some of the original papers on the subject and many of the references have not been consulted or verified by the writer on account of their inaccessibility; but what he has attempted is to fully consider the present series, and to amplify the necessarily incomplete conclusions based thereon by reference to already published cases. A list of the latter, now amounting to 328, will be found in the appendix.



The writer wishes here to express his thanks to Professor Keith, Curator of the Museum of the Royal College of Surgeons; to the Curators of the Museums of London, St. Bartholomew's, St. George's, Guy's, Great Ormond Street, St. Mary's, King's College, Middlesex, and Westminster Hospitals; to the Hon. Staffs of the same for permission to use their cases; and to Miss Caven for her help with the illustrations.

The subject is dealt with under the following headings:—

- I. The Writer's Series of Cases (p. 144).
- II. History, Literature, Frequency (p. 162).
- III. Morbid Anatomy (p. 163).
- IV. *Ætiology* (p. 181).
- V. Symptoms (p. 198).
- VI. Diagnosis (p. 199).
- VII. Prognosis (p. 202).
- VIII. Treatment (p. 203).
- IX. Summary (p. 206).

Appendices—A. Notes of some cases of Stenosis which have survived for three months or longer (p. 207).

B. List of Operations (p. 208).

C. List of Recorded Cases (p. 210).

D. References (p. 213).

#### I.—THE WRITER'S SERIES OF CASES.

CASE 1.—Tight stenosis of duodenum.—R.C.S. Museum, Terat. sect., No. 545-21 (Fig. 1). This interesting specimen was presented to the museum by Dr. Ivy Mackenzie. The writer has not been able to obtain any clinical details, but it is obvious that a gastro-enterostomy has been performed, and the anastomosis appears to have acted satisfactorily. That part of the duodenum above the biliary papilla is dilated to a spherical cavity whose only outlet is the narrow passage through which a glass rod has been passed. The specimen is figured as seen from behind, so as to show the entrance of the bile and pancreatic ducts. These open close together, in fact, practically by a

common orifice at the farther end of the stenosed portion. In the fresh specimen their orifice could only be seen with great difficulty, as it was in so narrow a part; but the specimen has altered slightly (in consequence of removing a piece for section), so that now the orifice is more obvious. The narrow part is about a quarter of an inch long, and feels fibrous to the fingers. There is an apparent break in the peritoneal covering of it. The dilated part has thick walls, but the walls of the bowel below the stenosis are quite thin, and it is noteworthy that its lumen is of about the normal size, having been distended by meconium and to some extent probably by food also. In this case the pylorus is about normal, but in some similar ones it is very stretched, so that the demarcation between stomach and duodenum is quite indistinct.

RCS MUSEUM. TERAT. SECTION 545.21

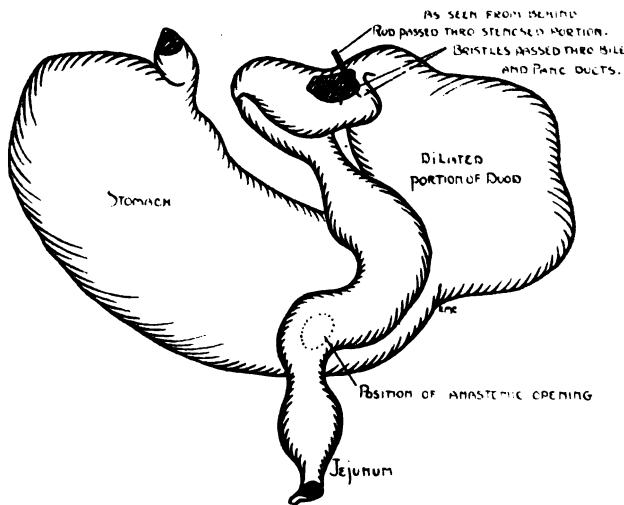


FIG. 1. (Reduced one-half.)

A *microscopic examination* of the stenosed part reveals a very interesting state of affairs. The mucous membrane, with its villi and Lieberkühn's glands, appears quite normal. The muscularis mucosa is well developed. In the greater part of the

length of the section (which is cut radially, parallel to the length of the bowel) there is no sign of Brunner's glands in the submucosa; but scattered through the muscular layer, in an irregular manner, are many glandular acini. One of these latter, in particular, is very large and lined by exceptionally tall columnar cells. Others are smaller with more cubical cells and not much lumen—more like Brunner's glands, but situated deeply in the muscular layer. One of these groups of glandular acini is, however, in the submucosa, though encroaching on the muscular layer. The muscular layer itself does not show the usual definite demarcation into a circular and a longitudinal coat, but the fibres run somewhat irregularly in groups, though, in the main, the circular fibres are aggregated nearer the submucosa. Unfortunately, some of the outer part of the muscular layer has been stripped off and lost in preparing the section, but it may be stated definitely that the muscular coat, as a whole, is of abnormal thickness. The glands referred to above are seen as far from the lumen as the section extends. Professor Keith, in reference to this specimen, says, "It is very common, especially in the foetal duodenum, to find Brunner's glands going right into and even through the muscular coat."

CASE 1a.—Atresia of duodenum.—R.C.S. Museum, Terat. sect., No. 545-2, presented by Ernest Shaw, Esq. (Fig. 2). This excellent specimen is shortly mentioned by Dr. Keith in his paper on "Congenital occlusions of the alimentary tract," and has also been recorded by Roe and Shaw. It is inserted here because it is a typical and simple example of this class of malformation. The early part of the duodenum is seen to be dilated to a sphere nearly as large as the stomach. This distension brings the first part of its "descending portion" more into a line with its "ascending portion," and consequently the normal relations are altered. The narrow portion of gut below the block (which is figured as sticking up for the sake of clearness) shows at its commencement the double orifice of the bile and pancreatic ducts (marked by a glass rod and a bristle), and is not so extremely contracted as in many specimens where the block occurs lower down, because in this case the bile can pass.

Roe and Shaw found the duodenal wall normal on section, but near the pylorus was the opening of a "large branched duct, lined by columnar cells." There were also irregular collections of gland tissue around. These were thought to be either an accessory pancreas or abnormal Brunner's glands, and recall somewhat the appearances in the previous case.

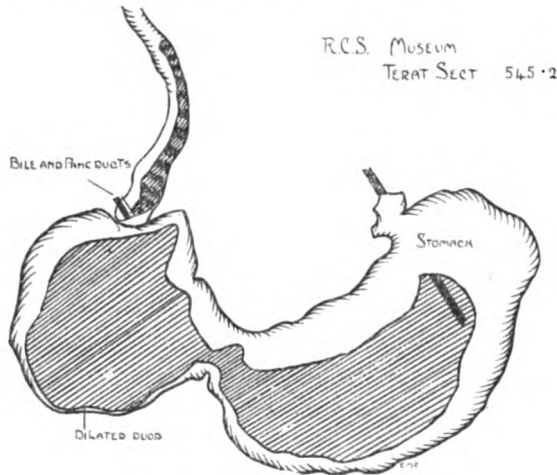


FIG. 2. (Reduced one-half.)

CASE 1b.—Atresia of duodenum.—St. Bart. Hosp. Museum, No. 3,635, E. 1, presented by Mr. J. Harrison. This truly extraordinary case is also mentioned by Dr. Keith in the above paper. The specimen is "from an infant aged nine months, who had been subject during life to recurrent attacks of vomiting. The vomit was abundant, the attacks occurring at intervals of some days. There had never been a normal evacuation of the bowels, the motions being constituted by small spherical masses of fæces. The stomach is enlarged, but otherwise normal. Immediately below the pylorus is a dilatation in the duodenum giving the appearance of a second stomach. This is rather smaller than the true stomach, and is limited below by a diaphragm passing inward from the whole circumference of the gut. There is *no evidence of an orifice in this diaphragm*, though

the history of the case proves that some opening must have existed." One can see no sign of the bile duct, but the septum appears to be at about the place where this should have entered the bowel. The gut below the septum is not "worm-like," but has apparently thin walls and a fair-sized lumen; this, coupled with the fact that there is no record of *bile* being vomited, makes it probable that the bile duct discharged its contents *below* the septum. A section of the latter examined microscopically shows it to be covered on either side with normal mucous membrane, beneath which is tissue continuous with the submucosa of the bowel wall. It also contains a layer of circular muscular fibres. No trace of scar tissue can be seen. Dr. Keith says, in reference to this case, "Sir Everard Home found that absorption from the stomach took place in the dog after the duodenum had been tied; in the case of this child absorption was apparently sufficient to keep it in life for nine months." The whole of the obstructing diaphragm is not present in the specimen, but what there is contains not the least trace of a healed orifice. Also, the section (cut across the removed part) showed no scar. Other instances of alleged prolongation of life with a complete alimentary imperforation will be alluded to later (page 202).

CASE 2.—Atresia of duodenum.—An infant, aged three weeks, was admitted into Great Ormond Street Hospital on September 29th, 1902, under Dr. Batten. There had been vomiting about ten minutes after food since birth, and also progressive wasting. The vomit was considerable in quantity, with much green mucus. The bowels had been moved, only meconium being passed. The child died on the day after admission. The stomach was found to be much dilated, as was also the duodenum, which was found to end abruptly at the point of entrance of the bile duct. The latter was dilated, admitting the head of a probe. The stricture was impermeable to air and water. The stricture cut firmly, and seemed to be of a fibrous nature. The stomach and duodenum were filled with green fluid with some curds. The intestine below the obstruction was collapsed and contained dark green mucus.

It is impossible to discover from the post-mortem record of this case whether the bile duct opened into the upper or lower segment of bowel. In either case it is hard to explain the presence of biliary matter both above and below the occlusion. One must consider the possibility of an accessory bile duct (q.v.i.).

CASE 3.—Stenosis of duodenum.—Guy's Hosp. P.-m. Reports, 1906, 447, Dr. L. E. Shaw. The infant was a male, and aged 14 days. There had been vomiting from birth, and very little had passed per anum. There had also been peristalsis from left to right, visible through the abdominal walls. The diagnosis was congenital pyloric stenosis. The stomach and duodenum were found to be almost identical in shape and size. The stenosis was in the neighbourhood of the duodeno-jejunal flexure, was slit-like, and scarcely admitted a fine probe. Presumably the bile duct opened well above it.

CASE 4.—Atresia of ileum.—The infant was admitted into Guys' Hospital under Mr. C. J. Symonds in March, 1905. The patient was an ill-nourished child of 5 days old. There had been continuous vomiting since birth. One motion was said to have been passed. This is described as not greenish, and slimy—probably only mucus. There was well-marked distension of the abdomen. The little finger could be passed through the anus, but experienced some resistance in the rectum. Firm pressure, however, enabled nearly the whole length of the little finger to be inserted, and the tip of this could be felt through the abdominal wall in the left inguinal region. Small soap, saline, and glycerine enemata were tried, but were at once returned. A catheter was passed per anum, but could be got up no higher than the finger. No trace of meconium was seen either in the returned enemata or on the finger. Intestinal occlusion above the rectum was diagnosed, and it was decided to explore, though but faint hopes of recovery were entertained. Upon the abdomen being opened by Mr. Ralph Thompson (in the absence of Mr. Symonds) the bowel was found to end bluntly in a large cul-de-sac, which was fixed with a few stitches to the abdominal wall, and drained with a small Paul's tube. Much

dark-looking matter at once came away. The operation could not be concluded as rapidly as was desired as some difficulty was experienced in replacing into the abdomen the coils of very distended bowel; and in spite of the very vigorous exhibition of stimulants and saline infusion, the child died eight hours afterwards.

*At the autopsy* a complete septum was found across the ileum a few inches above the ileo-cæcal valve. The septum was covered on either side by mucous membrane, and no trace of any peritoneal adhesions was found. All the small and large intestine below the obstruction was contracted and very small, being of about the diameter of a goose quill. The rectum appeared normal as far as the finger had been passed up, but the sigmoid above this was of the same calibre as the rest of the contracted intestine. No meconium was present below the obstruction. No other congenital defect was found. The contracted bowel could be distended by water pressure, and then appeared almost normal.

CASE 5.—Atresia of ileum (Fig. 3).—The infant aged 4 days, was admitted into the Hospital for Sick Children, Great Ormond Street, on April 1st, 1900, under Mr. Kellock. There had been continuous vomiting, both after food and also when no food had been given, since birth. The ejected matter had been brown and very offensive for the last 24 hours. The abdomen was distended. Laparotomy was performed, and the distended coils of small intestine traced down to an obstruction in the ileum. The bowel was drained with a Paul's tube, but the child died soon after.

*Post-mortem*, the bowels were very distended, and so injected as to appear almost black. The ileum came suddenly to an end three-quarters of an inch above the cæcum. A short fibrous band, about a quarter of an inch long, connected the blind end with the bowel below. The bowel above the obstruction was filled with gas and meconium, and the large gut below is also said to have had a little meconium in it. The mesentery at its attachment to the bowel was filled with blood. The large intestine was apparently normal in structure. At the point of stricture there was a break in the peritoneal covering of the bowel, which was not continued over the fibrous connecting band.

*Congenital Intestinal Occlusion.*

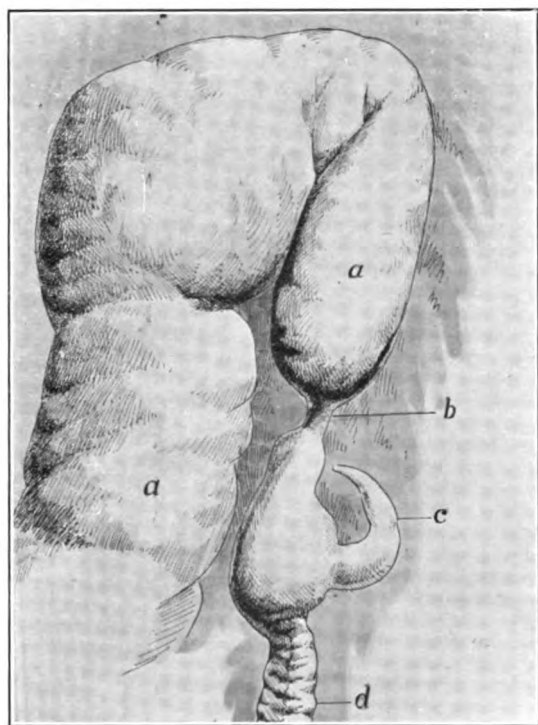


FIG. 3.

*a.* Dilated lower ileum. *b.* Fibrous band at seat of obstruction.  
*c.* Appendix. *d.* Colon.





CASE 6.—Atresia of ileum (Fig. 4).—R.C.S. Museum, Terat. sect., No. 550a. "From a female infant, aged 13 days, who was admitted (to hospital) on the 4th day after birth for constant vomiting and constipation. Enterostomy was performed, and the following day some meconium was passed per anum, which was probably derived from the intestine below the obstruction. The patient became much emaciated, and gradually sank."

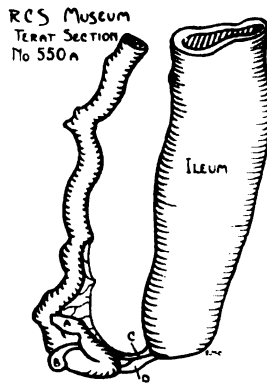


FIG. 4. (Reduced one-half.)

As the specimen shows, there is a complete interruption of the ileum (?), which is stated to be 50 m. below the pylorus. The diameters of the bowel above and below the block are one and a quarter and a quarter of an inch respectively. It is hard to determine the precise relation of the parts. The specimen certainly gives one the impression that the bowel has been ruptured. The microscopic appearances do not give much help. Two small fragments have been removed for section. That from B consists of fibrous tissue only. The piece from D is disorganised, and does not take the stain. In places there is an appearance as of muscular fibres, but not definite enough for any dogmatic statement to be made.

CASE 7.—Atresia of ileum.—R.C.S. Museum, Terat. sect., No. 550d. B. G. A. Moynihan, Esq., 1906. The new-born infant suffered from absolute constipation and vomiting. The

rectum and anus were found to be normal. An ileo-colic anastomosis was made, but the infant died 40 hours later. The specimen consists of the greatly dilated lower end of the ileum which is only connected to the cæcum by a small isthmus of tissue about a quarter of an inch in diameter. The appendix is of normal length. The colon is only about a quarter of an inch in diameter, and no communication can be traced between its lumen and that of the dilated ileum.

CASE 8.—Atresia of ileum (Fig. 5).—London Hosp. Museum, No. 1,164, Mr. Roxburgh. This interesting specimen, from an infant 5 days old, shows a complete stricture of the ileum at a point just above the attachment of a vestigial Meckel's diver-

LONDON HOSP. MUSEUM 1164

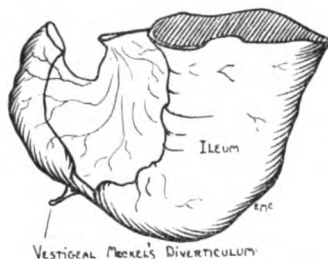


FIG. 5. (Reduced one-half.)

ticulum, and 12 inches above the cæcum. As in the other specimen of this nature (London Hospital Museum, No. 1,164) it will be noted that the constriction is not just opposite the attachment of the diverticulum, but rather above it.

CASE 8a.—Stenosis of ileum.—London Hosp. Museum, No. 1,164a. The subject from whom this specimen came was a youth of 19 years, who died of chronic intestinal obstruction (under the care of Mr. Waren Jay). The specimen is labelled "Congenital constriction of ileum, just above the point of attachment of Meckel's diverticulum." The latter had become invaginated, and so caused an intussusception 8 inches above the ileo-cæcal valve. The total length of the small bowel was 12 ft.

The peritoneum over the constriction is rough, as if from adhesions; and it appears to the writer that the narrowing is more probably acquired than congenital. One can conceive that it may be due to a chronic inflammatory change spreading round from the attachment of the invaginated diverticulum. This case is, however, included here because of the interesting association of stenosis and diverticulum. If the latter has any causal relationship to the former in this case, one can picture the same thing occurring in intra-uterine life, and, further, the invagination disappearing and leaving the stricture.

CASE 9.—Atresia of the jejunum.—M. H., aged 1 day, was admitted to Guy's Hospital under Sir (then Mr.) Henry Howse in November, 1900. The report of this case is very scanty, so that memory alone has to be relied on for some of the details. There had been vomiting since birth, and no motion had been passed. A finger was inserted per rectum, and it was believed that "a fibrous mass," or obstruction of some sort, could be felt one inch up the bowel. This was "partially broken down," to use the words of the report. It was considered possible that the gut might have been ruptured. The next day, as vomiting of slimy green material continued, the abdomen was explored, and the "colon was found to be undeveloped." A distended coil of gut was therefore fixed in the wound and opened. The child did well at first, but later sank, and died 10 days after the operation.

*At the autopsy* there was some purulent peritonitis, but not generalised. The small intestine was found to end in a blind cul-de-sac 29 inches below the pylorus. The enterostomy wound was an inch or two above this. All the intestine below the obstruction is described as being the thickness of a clay pipe stem. There was no communication whatever between the distended and collapsed part, though the latter was pervious right up to the obstruction. There was no trace of any old peritoneal adhesions over the site of the obstruction. There was no other congenital abnormality, except that the foramen ovale was found to be widely patent.

CASE 10.—Atresia of ileum.—St. Mary's Hosp. Museum, No. 857. Specimen from an infant who died a few days after birth. There is a block in the ileum, apparently quite complete, and probably its situation corresponds to the point of attachment of Meckel's diverticulum, though the exact site is not stated. The gut above and that below the block show the usual characteristics.

CASE 11.—Atresia of ileum.—A female infant, aged 3 days, was admitted into Guy's Hospital in December, 1910. (Med. Rep. 747a.) At birth it was a well-developed child, and a little faecal matter was said, by the nurse, to have come away per rectum when the infant was 22 hours old. At 48 hours vomiting commenced and continued till death. For 12 hours previous to admission the child had passed blood per anum, which latter felt normal to the finger. There was abdominal distension. Enterostomy was performed, but the child soon sank.

*Post-mortem.*—A few inches above the ileo-cæcal valve the bowel was only represented by a fibrous cord some inches in length. The bowel above contained bloody fluid (some of which had come away through the Paul's tube), and there were many submucous ecchymoses. There was also some hæmorrhagic extravasation into the lungs. The foramen ovale was patent, and other organs healthy. The gut below the obstruction was small and collapsed.

CASE 12.—Atresia of ileum.—London Hosp. Museum, No. 1,163, Mr. Fenwick, 1901. The notes on the infant from which this specimen was taken are very scanty. The child, a female, had no stool, and upon a rectal examination being made, "the gut seems to the finger obstructed 2 inches above the rectum." The specimen is not very clear, but the obstruction can be seen about half an inch above the cæcum. The gut above and below presents the usual characteristics.

CASE 13.—Atresia of ileum, with absence of segment.—St. Bart. Hosp. Museum, No. 3,635e. A full term, well-developed female infant, passed meconium in small amount on the first day, but none afterwards. Vomiting began on the second day, was bile-stained, and continued till death on the eighth day. "The

gut ends blindly at a spot two feet above the ileo-cæcal valve, and begins again after an interruption of fully an inch." In this interval there is simply a very thick, free edge to the mesentery. The bowel above is dilated to above the size of adult intestine. Below the interruption it is of about the diameter of the little finger, and not firmly contracted or worm-like. Both the condition of the bowel and the fact that the infant was able to pass meconium on the day of birth strongly support the view that this was quite a late break in continuity, probably after the sixth month when the bile is supposed to reach the lower ileum. There is no other abnormality, and no sign of peritonitis.

CASE 14.—Atresia of ileum, with loss of continuity of bowel. —D. L., aged 4 days, was admitted to the Hospital for Sick Children, Great Ormond Street, under Mr. F. J. Steward, on October 3rd, 1904. There had been no motion since birth, and also continuous vomiting of black-looking material. The anal canal would admit the tip of the little finger, and was found to end blindly about three-quarters of an inch up. Above this a protruding, rounded, rather hard mass could be felt, which bulged downwards when the child cried. This mass was explored with a trocar and cannula without result. An incision was also made backwards from the anus without finding the rectum. Later this incision was enlarged, when, after considerable difficulty and loss of much blood, an open end of the bowel was found and stitched to the margin of the wound. This bowel contained no meconium, and was contracted so that it was very difficult to stitch.

The infant died in five hours. At the post-mortem 7 ounces of blood were found in the peritoneal cavity; 39 inches below the pylorus the bowel was found to end in a blind sac, the size of a small goose egg. Lying coiled up on the inner side of the cæcum was what looked like an extra appendix, 6 inches long; this proved to be the lower end of the small intestine which was completely separated from the distended upper portion. There was no trace of any connection between the two

blind ends, the peritoneum having a free edge. The large intestine was collapsed and of small diameter, containing a little turbid mucus only. According to Treves, the small intestine of a new-born infant is 9 feet long. If this be so, a considerable segment of gut must be missing in this case. However, it is classed as an ileal obstruction.

CASE 14a.—Obstruction due to an abnormal arrangement of the mesentery.—M. W., aged 39 hours, was admitted into the Evelina Children's Hospital on October 9th, 1904, under Mr. C. H. Fagge. No normal motion had been passed, and there had been vomiting of greenish-brown material since 15 hours after birth. On admission the infant was in a very bad condition, with a distended abdomen, and almost imperceptible pulse. A finger was introduced  $1\frac{1}{2}$  inches into the rectum, which felt normal. The abdomen was opened in the left inguinal region, and, as nothing like normal large intestine could be found, a coil of dilated small intestine was opened and drained with a Paul's tube. Much thick meconium escaped. The child died shortly afterwards.

*Post-mortem*, the bowel was found to have been opened near the middle of the ileum. Above the opening the gut was almost empty, but below it was distended with meconium. The mesentery was absent from the last  $1\frac{1}{2}$  inches of the ileum, and this and all the large intestine were retro-peritoneal. At the point where the peritoneum became absent, the bowel diminished in diameter from  $1\frac{1}{2}$  inches to half an inch. About 4 inches above the ileo-cæcal valve the small intestine had ruptured. The large intestine contained only soft, white, cheesy material. There were no signs of foetal peritonitis, nor was any septum or valve found at the site of obstruction, there being no real occlusion.

CASE 15.—Multiple occlusions of ileum (Fig. 6).—R.C.S. Museum, Terat. sect., No. 550, F. Fawson, Esq., 1883. Specimen from a child who "died on the fifth day with faecal vomiting. The rectum was also apparently obstructed two inches above the anus." The infant was said to be otherwise well developed. This is a good, if rather extreme, specimen of this

class of multiple occlusions. From above downwards we may note:—*a*, The walls of the duodenum so stretched as to be thrown into overhanging folds, the lumen getting more and more dilated from above downwards, till at its lower end it is almost

RCS MUSEUM TERAT SECTION 550

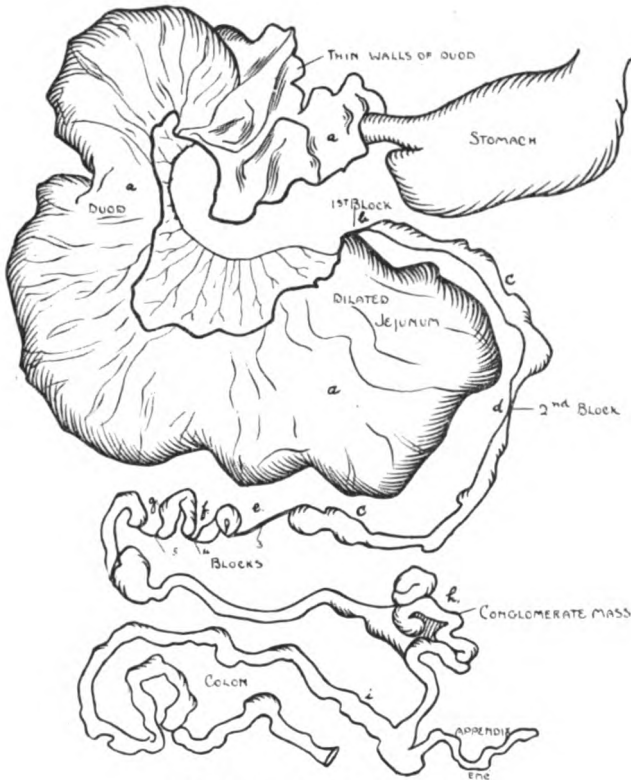


FIG. 6. (Reduced one-half.)

double the diameter of the stomach; *b*, The bowel quite suddenly being reduced to the size of a thread; *c*, Contracted bowel following on this block; *d*, *e*, *f*, and *g*, Further blocks in the lumen; *h*, Conglomerate worm-like coils; succeeded by *i*, The contracted "large" intestine. In some cases where the bowel is



not quite blocked (probably), it is still extremely narrow, and all stages of development can be seen. The obstruction in the rectum referred to above was probably only due to the narrow contracted bowel.

CASE 16.—Multiple atresia of jejuno-ileum (Fig. 7).—R. K., aged 5 days, was admitted to Great Ormond Street, under Mr. W. Arbuthnot Lane. On admission the infant was moribund, with faecal vomiting. This child was said to have passed seven stools, all whitish yellow, one of them containing a little blood. The abdomen was hurriedly explored, and the infant died next day.

*At the autopsy* the bowel was found to be cut up into divisions, with intervals containing a thickened free edge of mesentery in place of the gut. The first break was 10 inches below the pylorus and 2 inches long; then came 3 inches of bowel, followed by a half inch gap; to this succeeded 6 inches of gut, with yet another break. In other parts the bowel was very much constricted without being quite absent, so that there were different degrees of development present. The interesting specimen from this case, as also that from Case 4, may be seen in the museum at the hospital. A similar case to that just related, only showing even more points of constriction and occlusion, has been reported by Emanuel.

CASE 17.—Multiple stenosis of jejunum.—King's Col. Hosp. Museum, Dr. Still. This specimen, from a female infant aged 5 days, shows an extremely dilated duodenum, so much so that it must have filled most of the right half of the abdomen. The first 2 or 3 inches of the jejunum is likewise dilated, but it then suddenly narrows to one-eighth of an inch in diameter; half an inch further on it is still further contracted, and looks as if completely occluded; but the writer was able to pass a bristle through this second constriction. The bile and pancreatic ducts were found to be normal. There were numerous easily separable adhesions on the great omentum. After the second constriction the worm-like contracted bowel soon enters into a conglomerate mass with adherent coils which has not

*Congenital Intestinal Occlusion.*

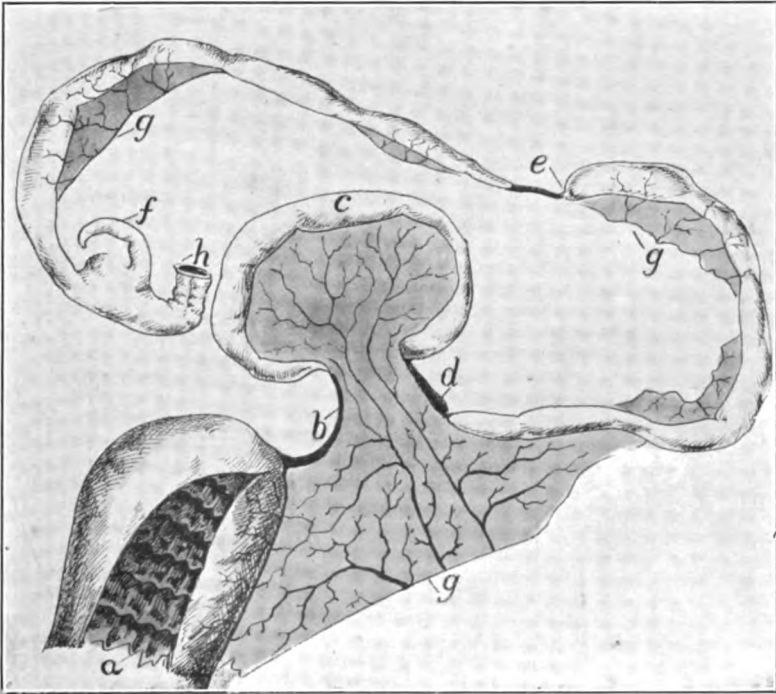


FIG. 7.

- a.* Dilated ileum cut open. *b.* First break in continuity of bowel.  
*c.* Contracted bowel. *d.* Second break. *e.* Third break. *f.* Appendix.  
*g.* Cut edge of mesentery. *h.* Contracted colon.



been unravelled, or doubtless other constrictions would be brought to light. From this mass the "large gut" (still worm-like), can be traced to the rectum. The last 2 inches of rectum are more distended, probably by digital examinations or injections per anum. The other abdominal organs are normal.

CASE 18.—Double constriction of jejunum.—St. George's Hosp. Museum, Series IX., 71k. Clinical notes. Female, 3 days old at death. The bowels were said to have acted once, but the nature of the stool is not stated. After that there was no further motion: vomiting set in, and distension ensued. Sir W. Bennett performed enterostomy, but the child only survived two hours. There is great narrowing of the jejunum at a point three feet below the pylorus, and one c.m. further on it becomes still further reduced, the following bowel being the size of a goose quill. Through the second constriction neither a probe nor air can be made to pass. The goose quill bowel continues down to a little above the anus where it is large enough to admit the little finger, which was doubtless the dilating agent.

CASE 19.—Double atresia of ileum, with absence of segments.—St. George's Hosp. Museum, Series IX., 71a. The specimen is from a young infant with imperforate rectum. A trocar was passed up the anus, but without relief. *Post-mortem*, complete absence of two portions of the ileum was found, there being a thread-like connecting band in the gaps. There is an abnormally long appendix.

CASE 20.—Extreme form of mal-development of the intestines.—R.C.S. Museum, Terat. sect., No. 550c, Dr. Huggins. This specimen is labelled "Intestine of foetus; it is blind at either extremity, and forms a much convoluted mass." It consists of a conglomerate mass of ill-developed, worm-like bowel. One cannot trace the difference between small and large gut. It is impossible to say without unravelling the specimen the number or extent of the occlusions, as the coils are adherent to each other in many places, and show obvious signs of foetal peritonitis, especially at the upper part. No clinical history is appended.

CASE 21.—Atresia of colon (Fig. 8).—London Hosp. Museum, No. 1,165, Mr. Rivington, 1882. The clinical details, as far as can be ascertained, are as follows: The infant, who died when 4 days old, had had no stool, and had constantly vomited. The vomit latterly was "fæcal." The child was found to have an imperforate anus, and was operated upon for that, but died the next day. *Post-mortem* the lower ileum, cæcum, and appendix were found to be very greatly distended. The com-

LONDON HOSP. MUSEUM. 1165

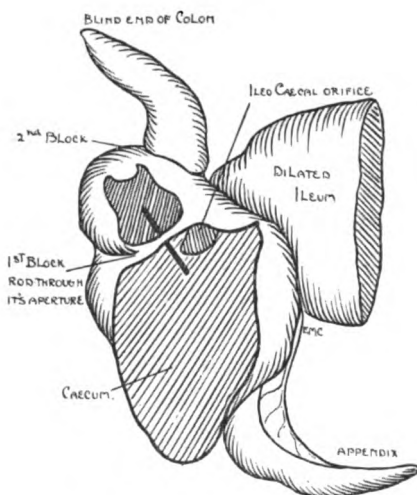


FIG. 8. (Reduced one-half.)

mencement of the ascending colon is obstructed by a valve, but not quite completely, as a small hole perforates the partition (a rod is represented as being put through this orifice). Above this valve is another dilated part the size of a walnut; then follows a second constriction, and yet another dilated part. This latter is a blind sac, as beyond its upper extremity there is no sign of the gut, which presumably was absent as far as the anus. This specimen is in an excellent state of preservation.

A section of a fragment of the septum removed by the writer shows two well-developed muscular coats (a circular and a slightly thinner radial), covered on either side by mucous membrane without glands.

CASE 22.—Occlusion of colon.—Guy's Hosp. Museum, No. 772. From a male infant who died when seven days old. Nothing was passed per anum. A finger passed up the rectum "seemed to be stopped by a septum." The next day the whole length of the finger was inserted. Enterostomy was performed, the small bowel being opened, but the infant died the day after. The membranous septum seen in the specimen lay by the splenic flexure, and completely occluded the bowel 9 inches above the anus. There was an unusually large ductus arteriosus. N.B.—This specimen is mentioned by Dr. Hale White in Allbutt's System of Medicine.

The microscopical examination of a small fragment of the septum, removed by the writer, shows that it contains two well-marked layers of muscle (circular and radial), covered on either side by mucous membrane containing but few glands very irregularly distributed.

CASE 23.—Atresia of colon.—R.C.S. Museum, Terat. sect., No. 550b. C. Maisey, Esq., 1895. The specimen consists of the greatly distended colon of an infant ending blindly. The rectum is absent. There is no clinical history.

CASE 24.—Obstruction of colon by adhesions.—Guy's Hosp. P.-m. Reports, 1899, No. 386. "The descending colon, with its meconium, formed at its lower end a pouch, from which a small tunnelled orifice, half an inch in length, leads to a large, thick-walled cavity, apparently the sigmoid or rectum." This cavity contained pinkish fluid, but no meconium (nor were its walls stained with meconium); it was distended so as to reach above the umbilicus, and had been drained apparently by a perineal incision made to relieve an imperforate anus. A tough white fibrous band crossed this cavity, and the bladder was adherent to it. There was also a double hydronephrosis. It is almost impossible from the above description, or from an examination of

the specimen, to state clearly the exact relationship of the parts; but this case does definitely prove the existence of a foetal peritonitis. Here we find it chiefly pelvic in position, interfering with the sigmoid and obstructing the ureters also.

## II.—HISTORY, LITERATURE, AND FREQUENCY.

Probably the first case recorded was that of Osiander in 1797. He was soon followed by Voison, Lassus, and Aubery, the former of whom, in 1804, performed enterostomy for this condition. Meckel mentioned several cases of the kind in his pathological anatomy of 1812, and also put forward his views as to their aetiology. Sir J. Y. Simpson published his paper on foetal peritonitis in 1838, and therein mentioned some cases of intestinal atresia. Other odd cases were recorded, but nothing exhaustive on the subject appeared till 1877, when Theremin collected all the then known cases, and entered elaborately into the subject, more particularly from the pathological side. His figures as to the frequency of the affection are still quoted. He says that in 150,000 children at a foundling Hospital at St. Petersburg, there were only nine cases, and only two in 111,000 at a similar hospital in Vienna. Silbermann collected 57 cases in 1882, and Gartner contributed a paper the next year. In 1889 Hudson showed his specimens of the condition at the Pathological Society, and Bland Sutton published his views as to the aetiology of the subject. Schlegel, in 1891, collected 89 cases. The first paper of any size in English was published by Louise Cordes in the United States in 1901, but deals only with the duodenum. She gives notes of 57 cases of obstruction on this situation. About the same time appeared papers by Braun and Bretschneider. The most exhaustive collection is that of Kuliga, published in 1903, where the total number of cases rises to 189. Savariaud made a smaller collection the same year, and says that 44 of them had been treated surgically. A paper by Clogg appeared in 1904, based on two cases of his own, but with free reference to the literature.

As regards frequency, Theremin's figures indicate that this defect occurs in from 1 in 16,000 to 1 in 55,000 infants. Trélat says that our ano-rectal imperforation occurs in 1 out of 10,000 births. Braun calculates that of those born in Germany in 1899 (rather under 2,000,000) probably 118 had a congenital occlusion of the bowel. This figure, though very hypothetical, gives some indication of the number of these cases which must die undiagnosed, or, at least, which are never put on record. It may be that the condition is not so very much rarer than imperforate anus as one might expect: the one affection being so obvious cannot be missed, the other most certainly is not so constantly in the mind of the practitioner and not so obvious, hence it often is missed.

Several writers have reported more than one case. Bland Sutton has seen three at least. Durante saw six in one year at the Maternité. On the other hand, several physicians and surgeons of large experience tell the writer that they have never yet seen a case of this kind.

More seem to have been noted, in proportion to the population, on the Continent than in England. This is doubtless because post-mortem examinations are more universally made there.

As stated above, reference will be found in the appendix to 328 cases. Those cases in which there was obstruction, but no definite anatomical narrowing or block, have been excluded as far as possible, though in many cases one cannot find out the exact condition.

### III.—MORBID ANATOMY.

1. Congenital intestinal occlusion is most often *single*, as in 17 out of the above series of 24. Of the multiple cases, two (18 and 19) had two strictures only; two had three (16 and 21); another, five (15); and in two others the number of constrictions cannot be definitely stated (17 and 20).

In a larger series it is found that in five cases out of every six the atresia is single; that in the multiple cases there are generally more than two occlusions; and that most of the multiple cases occur in the jejuno-ileum.



2. The occlusion is most often *complete*, as in all the writer's series, except Cases 1, 3, and 8a (probably not congenital). In cases of multiple constrictions it is very usual for some of the occlusions not to be complete, i.e., stenoses and atresias co-exist. Pure stenoses are found to comprise about one-tenth of all cases. This includes many found in adults, which are not certainly congenital, and which comprise about half of the stenoses.

No. 14a (Mr. Fagge's case) in which the obstruction was due to a kind of kink caused by an abnormal arrangement of the mesentery, though there was no real occlusion or stricture of the bowel, may be compared to those cases in which the block occurs at the duodeno-jejunal flexure, and which may be due to developmental changes in the mesentery at that point (q.v.i.).

Durante has recorded a case in which the end of the duodenum was kinked over a band without any real occlusion. These cases are of great interest in connection with Arbuthnot Lane's well-known views about kinks in the jejuno-ileum, as it is in the above situations that he says they most often are demonstrated.

In the appendix will be found short notes of the cases of stenosis which survived three months or more. These, though much rarer than the more complete occlusions, are of far more importance as regards treatment, being more amenable to surgery.

3. *Position of the Occlusions.*—Of the 24 cases, 17 were single and 7 multiple. Of the 17 single cases: 3 were in the duodenum (1—3); 1 was in the jejunum (9); 10 were in the ileum (4—8, 10—12, 13 and 14); and 3 were in the colon (22—24).

The duodenal obstructions were, in Cases 1 and 2, at the point of entrance of the bile duct; in Case 3, at the duodeno-jejunal flexure. (N.B.—Occlusions in this situation are in this paper classed as duodenal.)

Of the 10 single occlusions in the ileum, 3 were within 2 inches of the ileo-cæcal valve (5, 7, and 12); 3 more were within a foot of the valve (4, 8, and 11); that is 60 per cent. were within

one foot of the valve. In Case 10 also most probably the *lower* ileum was affected. The three single obstructions in the colon were all (most probably) in the descending portion.

Of the 7 multiple cases, 1 was a double occlusion of the jejunum (18); 1 was a double occlusion of the ileum (19); and 1 was a treble occlusion of the colon (21). 4 were situated in the jejuno-ileum, and had three or more blocks.

In a larger series it is impossible to classify accurately, as in many cases one cannot ascertain the position of the occlusion; for instance, quite often one cannot gather whether the defect is in the jejunum or ileum. Many cases are described, particularly in the older papers, as being at the ileo-cæcal junction. The writer considers, from evidence afforded by his series, that many of these may have been in the ileum quite close to the cæcum (as in 5, 7, and 12). Very few cases have been noted just on the other (distal) side of the ileo-cæcal valve, at the junction of the cæcum with the colon; therefore, in the figures given below, these few have been put into the same group as the lower ileal and ileo-cæcal obstructions. With these premises the recorded cases may be roughly grouped as follows:—

*Duodenum—*

Above biliary papilla	...	...	29	} 92 or 28 per cent.
Opposite " "	...	...	17	
Below " "	...	...	21	
At duodeno-jejunal flexure	...	...	15	
Unclassified	...	...	10	

*Jejuno-ileum—*

Unclassified ...	...	...	55	} 124 or 38 per cent.
Lower ileum, and in the immediate vicinity of the ileo-cæcal valve	...	...	69	

<i>Colon</i>	...	...	...	...	20 or 6 per cent.
<i>Unclassified</i>	...	...	...	...	28 or 8 per cent.
<i>Multiple Occlusions</i>	...	...	...	...	64 or 20 per cent.

Total .. ... 328

One sees that the proportions come out rather differently from the small series of the writer, the duodenal obstructions forming rather more than one-fourth of the whole, and single occlusions in the colon forming only 6 per cent.

4. *The Occlusion itself* may be in several forms, as shown diagrammatically in Fig. 9:—

DIFFERENT TYPES OF OCCLUSION

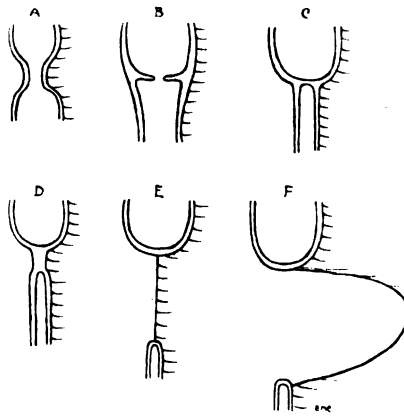


FIG. 9. (Reduced one-half.)

- A. Simple narrowing, more or less marked (Case 1, Fig. 1, and Case 8a).
- B. Perforated diaphragm (Case 21, Fig. 8).
- C. Complete diaphragm (Cases 4, 9, 21, and 22).
- D. Short band, connecting the ends of the bowel (Case 5, Fig. 3; Case 6, Fig. 4; Case 7; Case 8, Fig. 5).
- E. Thread-like band, along the free edge of the mesentery, the ends of the bowel being some distance apart (Cases 14, 15, and 20).
- F. Gap in mesentery also, there being no direct connection between the ends of the bowel, as shown in Fig. 10 (Keith).

It is impossible to give figures of the relative proportions of these types, but it may be stated that A, B, and F are rare forms; and that, of the others, some kind of band is three times as frequent as a diaphragm.

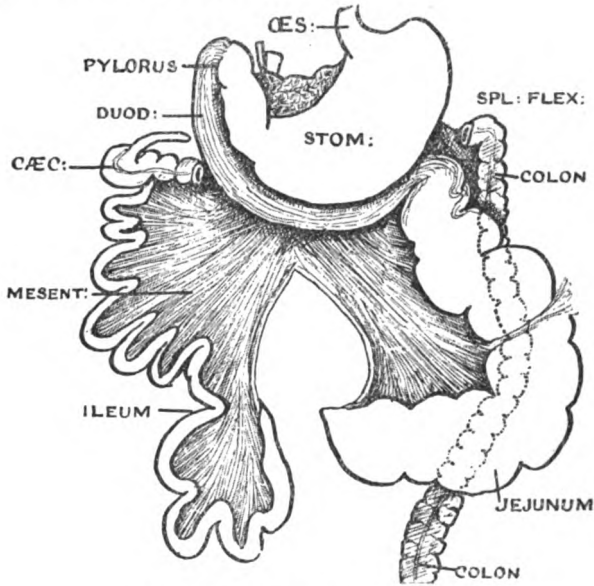


FIG. 10. (Reduced one-half.)

*Microscopical Appearances.*—As regards A, the writer can find no record of a section of a slight narrowing. The appearance of a section of a more marked duodenal narrowing is alluded to under Case 1, where the usual coats were present, but somewhat irregularly arranged, and with some anomalous glandular appearances, as also in Case 1a. Mohrmann, in a more extreme case, supposed to be caused by the head of the pancreas, found a modified mucous membrane with no definite submucosa or muscularis mucosæ, and no Brunner's glands. Carling, in a duodenal stenosis in a woman of 47, found no evidence of inflammation and no sign of a cicatrix; and Rolleston, in a jejunal stricture in a man of 25, found no evidence of inflammation or tubercle.

B and C.—It is more convenient to consider these together. A section of a fragment removed from a perforated diaphragm in an adult (Silcock's case) by the writer, showed from one surface to the other:

1. Mucous membrane with villi and Lieberkühn's glands.
2. A sparse layer of circular muscular fibres which was absent in some places.
3. A well-marked layer of radially running muscular fibres.
4. Very loose connective tissue.

Then followed similar layers to the above, but in the reverse order, viz.:—

5. Radial muscle.
6. Circular muscle.
7. Mucous membrane.

The septum corresponds, therefore, to a *re-duplication of the whole intestinal wall*, except that the submucosa (with Brunner's glands) is absent.

A similar fragment removed from Case 22 shows two well-developed muscular coats covered on either side by mucous membrane without glands. Also in Case 23 (complete septum), the appearance is similar to the above, except that there are a few glands.

A section of a duodenal septum in the case of Champneys and D'Arcy Power revealed that the circular muscular coat of bowel was prolonged into the septum (but not the longitudinal); it was covered on either side by normal mucosa and submucosa.

Buchanan found that the circular muscular coat sent a "few fibres into the mucous fold, of which the (duodenal) septum consists." Also, he found Brunner's glands normal above the block, but small and difficult to find below.

Cordes also notes in her case that there were few, if any, Brunner's glands in the neighbourhood of the papilla (where the block was), whilst in a normal child of the same age were found almost a continuous layer.

Wyss, in a duodenal valvular occlusion, found mucosa and submucosa only, whilst Baron and Valenta found mucosa alone.

D and E.—Kuliga found in his case (of stenosis and atresia) that in the stenosed part the mucous membrane was superficially eroded, with fresh hæmorrhages. In the submucosa were alveoli looking like lymph spaces, and filled with endothelial cells with large nuclei. These alveoli were also seen between the two muscular layers. The longitudinal muscular layer showed peculiar radially-running splits. In a fibre-like atresia he found both muscular layers present, and within these an excess of blood vessels. He concludes that the microscopic appearances do not explain how the epithelium has gone, but suggests that an overgrowth of blood vessels is the primary defect.

In Bretschneider's paper there is an exhaustive account of the appearances of a connecting band in the lower third of ileum. He found the mucosa absent, muscularis mucosæ weak, submucosa swollen and infiltrated, circular muscle weak, and longitudinal muscles well developed. In the centre of band were signs of hæmorrhage (pigment cells, etc.), signs of meconium (wool, hair, etc.), and giant cells. These were not like those of tubercle, and no bacilli were found in them.

Several others have confirmed the above appearances in these bands, that is, in general terms, the presence of the muscular coats, and absence, more or less complete, of the mucosa. Blood pigment was also found in the centre of the band by Martens and Tobeitz. (Other observers were Helmholtz, Bergallonne, and Wyss.)

In Case 6 the writer could not find definite signs of altered intestine in either of the bands (B, C, and D) which connect together the ends of the bowel.

*To sum up the microscopic appearances*, we may say, with regard to septa, that they are certainly not cicatricial, and that they consist of the usual coats of the bowel, including the muscular layer. This distinguishes them as not being simply hypertrophied valvulæ conniventes which contain no muscle. With regard to the "bands," they also show the normal elements of intestinal wall, but modified, and that chiefly near the lumen, as though the causative trouble has affected the mucous membrane most.


5. *The Bowel above the Obstruction* is typically very dilated, sometimes extremely so. For instance, in Fig. 6, the intestine is of about double the diameter of the stomach, and it is quite usual for it to be dilated to the size of normal adult bowel. When seen fresh it is also engorged with blood, and, therefore, of a dark colour, sometimes nearly black. There is often actual hæmorrhage into the bowel or stomach, as in Case 11, where many submucous ecchymoses were found. In this case also there was hæmorrhagic extravasation into the lungs, and blood had been lost per rectum. As there would not have been any engorgement of the bowel below the block, in this and other similar cases, we may take it that there is a general hæmorrhagic tendency. Emerson, Darier, and Hervey found black matter in the stomach of their cases of duodenal obstruction. Durante found blood in the stomach in a case of congenital valvulus of small gut and cæcum, and in many instances vomiting of blood or black matter is noted (q.v.i.). In Case 5 the mesentery at its attachment to the bowel was filled with blood.

The walls of the intestine are not only stretched, in some cases they are said to be hypertrophied also. One would hardly expect this, in complete cases, at least, as the gut has had no chance of overcoming the resistance, or of normally functioning. Davies-Colley, in reporting his case of occlusion by a complete septum a few inches above the cæcum, said, "Above the septum the wall of the intestine was much thickened." In Polaillon's case, he notes that the walls of the dilated cul-de-sac were 1.5 c.m. thick. Ducros says that there is hypertrophy of the walls of the bowel above in one-third of the cases, though one suspects that in many instances this so-called "hypertrophy" is due to serous infiltration or inflammation.

It is probable that the dilated part of the intestine is increased in length as well as in diameter.

The *content* of this dilated bowel is, of course, made up of the ingesta and of the meconium, which cannot pass away in a normal manner. These are often more or less mixed with blood.

In this connection allusion may be made to those cases in which bilious matter is present above a duodenal constriction (as shown by bilious vomit or found post-mortem) even when the latter is above the level of the entrance of the bile duct. Vomiting of greenish fluid, taken for bile, was recorded by Wilks, Hirschsprung, Hobson, Wyss, and Ferber, and Northrup and Anderson also found bile-stained mucus or meconium in the stomach. In all of these cases the block was definitely stated to be in the duodenum, above the biliary papilla. The reporters of these cases were at a loss to explain the phenomenon. Wilks says, "The fluid ejected must have been gastric secretion itself." Hirschsprung analysed the fluid and found bile pigment present, and concluded it was "incomprehensible." Hobson thinks it must have been "altered, exuded blood." Northrup surmised that the colour of gastric contents might be derived from the general circulation, but probably was nearer the mark when he suggested that a small branch duct might be present opening into the stomach. Louise Cordes, in her excellent paper, probably gives us the correct explanation. In her case there was demonstrated to be an abnormal branch of the common bile duct, or, rather, a double opening. The case was very like Case 1, the dilated upper part of the duodenum apparently ending blindly, but a narrow canal 4-5 mm. long was found to open into it, and a probe passed down this issued from the orifice of the common duct *below* the block, *i.e.*, the probe had passed into one orifice of the common duct and out of the other, so that these two openings of the duct formed the only communication between the dilated and contracted bowel. These branch ducts were microscopically found to be normal. Cordes suggests that in some of the above cases a similar accessory branch duct existed, which was overlooked, and which discharged bile *above* the stricture. Thus, in one of Therman's cases, speaking of the cul-de-sac of duodenum *below* the stricture, he says, "Into its upper portion the ductus choledochus opens, after uniting at an acute angle with the narrow canal which connects the two portions of the duodenum," and his other





case is similar. In these two cases green vomit is not mentioned, but one can see how it could be explained. From the figure illustrating Wilks' case, a somewhat similar state of things existed, there being a depression in the cul-de-sac on the gastric side of the constriction (not mentioned in the text), agreeing with Cordes's description of her case. Foster also says that the bile duct may divide, one branch opening into the stomach, or even into the large intestine.

In Crook's case of duodenal occlusion the pancreatic duct opened into the upper cul-de-sac, and its terminal part was dilated by the presence of the injesta, and one can see how the bile duct, similarly dilated, might appear to be the continuation of the bowel.

Another less likely explanation which has been advanced to meet these cases is that the green vomit is due to the foetus having swallowed liquor amnii containing some meconium previously passed.

If duodenal distension becomes extreme, the pyloric ring is obliterated, and one can distinguish with difficulty between the stomach and duodenum. The line of demarcation can, however, always be seen internally, the mucous membrane of the stomach being yellowish-white and opaque, and that of the duodenum being whitish-grey (Ducros).

In several instances the dilatation of the bowel has caused it to burst, causing either peritonitis or an abscess, as in Case 14a and in one of Helmholtz's cases.

*Connection with Hirschsprung's Disease.*—The dilatation of the colon observed in this affection is dependent in many cases upon a narrowing of some part of the colon or rectum. Okinczyk, in an interesting paper on this subject, tells of a case of a dilated transverse colon (in an adult) where a "segmentary atrophic atresia" was found in the descending colon. The arteries supplying the narrow part were very small. He had another case similar, and definitely states that the contraction was not due to spasm. Hartman also has written a paper on a case of congenital constipation consequent upon "atrophy" of the pelvic colon (q.v. Appendix A).

Treves also adheres to this view, and says (of idiopathic dilatation of the colon in children), "The great majority of them, at least, depend upon a congenital narrowing of the lower extremity of the large intestine."

Grisel follows the same line, and quotes Duplex as having seen a patient with Hirschsprung's disease who at birth had had an imperforate anus. Grisel also saw with Kermisson a patient, aged 3 years, with enormous distension of the colon, following an anal imperforation. This had been treated in the first place by simple puncture, and a later rectal examination showed a narrowing, the stretching (*débridement*) of which led to cure in some weeks.

6. *The Bowel below the Obstruction.*—The most outstanding feature of these cases, also the most important one from the point of view of treatment, is the condition of the bowel below the obstruction. This in no way compares with the empty gut found below an acquired obstruction, in that it is not merely empty and collapsed, but firmly contracted up, never having functionated. Thus, we find it designated "undeveloped," "of the thickness of a goose quill," similar to a "clay pipe stem," "worm-like bowel." The last term, perhaps, best describes its appearance, feel, and size. In most cases where there is a single obstruction, which is complete, anywhere below the biliary papilla, the worm-like gut extends from the obstructed point down to the anus, though often, perhaps generally, the last two or three inches above the anus are larger, having been dilated up by the examining finger, or by enemata. On the other hand, where the block occurs above the biliary papilla, the bowel below is distended to some extent by the bile, and therefore is more natural in appearance, as in Cases 1 and 1a. Also, in some other cases where the block has occurred late in foetal life, after the bile has distended the affected portion of gut, the lower part of the bowel is more nearly normal in calibre, as in Case 14.

In very rare instances only is there found any distension below a complete block, but this was recorded by Lyot, where the bowel

was very big and filled with meconium even below the obstruction. In the figure of Emanuel's case one sees that there is a less degree of distension below the first occlusion. This worm-like bowel is also probably shorter than the normal, and this fact, together with its small diameter, makes it occupy very little room in the abdomen. It is generally found lying against the spine and hidden by the distended coils.

Many instances are on record in which the ileum discharged its contents at the umbilicus. Cazin has noted, in two such cases, that the disused bowel below the fistula was contracted, and in his figures it looks typically "worm-like." Broca also has called attention to the same fact in similar cases associated with ectopia vesicæ. It would appear, therefore, that the extreme contraction of the gut was merely due to its non-distension by meconium.

A *microscopic examination* of such a worm-like bowel has been made by Bretschneider in his case of ileal stricture. The lumen ( $1\frac{1}{2}$  to 2 mm. in diameter) contained red blood corpuscles and mucin. Lieberkühn's crypts were more frequent on the mesenteric side of the bowel. The muscularis mucosa was weak, but traced all round. The submucosa was thick, poor in nuclei, and with full blood vessels. Some capillaries were seen to pass through its wall and open freely into the lumen. The circular muscle was feebly developed, and absent sometimes on the "convex side." The longitudinal muscle went all round, and was stronger. The serous layer was normal. The vessels of the mesentery were very full.


What we may call the "hæmorrhagic element" in this case would probably not be present in others, and we can say, speaking roughly, that all the normal coats of the bowel may be expected to be present in these cases, but poorly developed.

In Case 4 an attempt was made at the autopsy to dilate up some of the contracted bowel, and it was found that this could be done by water pressure, the gut then assuming an almost normal appearance. This procedure was also undertaken at the post-mortem examination in the case recorded by Polaillon, and

with a like result. Similarly, in these cases the rectum and sigmoid can be dilated up by the examining finger as far as one can reach, though some force is required, and the impression may be given that there is some real obstruction present in that part of the bowel. In Case 9 it was considered quite possible that the bowel might have been ruptured. In one case it is stated that the contracted bowel, when distended with air, was still only of a very small diameter. It is probable that air used with a syringe is not so good a dilating force as water from an ordinary tap, which was used to dilate up the bowel in Case 4. However, it is also certain that in some cases there is a real defect of development of the bowel apart from the places of complete obstruction.

If one examines Figs. 6 and 7 one sees that in some places there is great narrowing without actual obliteration, and in some of these multiple cases all stages of development may be seen in the same specimen. The personal view of the writer is that in most cases where only one obstruction exists, the "worm-like" bowel is suffering rather from want of distension than from want of development, but that in many cases of multiple occlusions there is often also a defect of development between the actual blocks. This view is rather confirmed by Savariaud, who says, "the most important role of the meconium is to distend the intestine."

*The content of this worm-like bowel* is typically a small quantity of colourless or greyish mucus entangling epithelial debris, and this is all that can generally be obtained by enemata in these cases. Though it is often noted that blood has been passed per anum (q.v.i.), it is very rare to find a record of its being discovered in the contracted bowel post-mortem. In a large number of cases meconium is noted as having been found in the contracted bowel or passed per anum. Thus, in Case 2, meconium had been passed, and dark green mucus was found in the contracted bowel post-mortem. In this case, however, it is possible that the bile duct entered the gut below the block. In Case 5 a little meconium was found in the bowel below, and



in Case 6 meconium was passed per anum. In this latter case the specimen certainly gives one the impression that the bowel has been ruptured, and that at no very distant date, which may have been after the meconium had descended past that point. In Case 13, also, meconium was passed on the day of birth, but not after. The bowel below the block in this specimen is much larger than is generally the case, and points to its having been distended by meconium. Probably this is another case of late obstruction. In Case 16 seven stools are said to have been passed.

In Emanuel's case a large motion of meconium of normal appearance was passed in hospital as the result of an enema, and yet there were five complete blocks (and other narrowings) high up in the small intestine. Also in this case the segment of bowel *after* the first block was distended.

Ducros found meconium below in 5 cases out of 35 of complete occlusion. He says that it was always small in quantity, and followed later by the usual greyish stools.

In Mohrmann's case meconium was passed, but no lanugo was found in it on microscopical examination. One would, of course, expect to find meconium below the block when the latter was not complete, or when it occurred above the entrance of the bile duct. Excluding these, however, material taken to be meconium has been found in the gut below the obstruction or in the stools by many reliable observers.<sup>1</sup> How can we explain these observations? Manifestly in two ways. The first assumes that "meconium" is a rough term, and that bile is not a necessary constituent of it. The second affirms that the obstruction must have occurred after the bile has passed that point in its descent down the bowel.

Meconium is understood to be the term applied to the dark-green fæces of the first three days or so of life, and before the appearance of the light orange or yellow milk fæces of the suckling. Meconium is usually held to consist mainly of bile (with its product cholesterin), but also contains other items which have been swallowed, viz., lanugo hairs, epithelial débris,

and vernix caseosa. Besides these there are the gastric and intestinal secretions (if any), and certainly mucus and more epithelial debris locally shed.

Foster speaks of meconium being found in the foetus, even when the liver is absent. Rolleston also says that in cases of congenital obliteration of the bile ducts the first motion is usually like meconium, but the subsequent stools colourless. He mentions the fact that in these cases green-coloured stools are said to have followed a dose of calomel, and to be due to a coloured compound of mercury and sulphur, or to ulceration caused by the calomel! Also, he says that the colour has been attributed to micro-organisms. As regards bacteria, Lesage claims to have isolated a special organism causing the green diarrhoea of children, but according to Garrod and others his results have not been confirmed.

Possibly the dark colour of the stools in some cases may have been due only to altered blood, and the motion designated "meconium" as a result of imperfect observation. One knows how often patients, and perhaps those with some medical knowledge, will describe matter ejected from the stomach as being bile-stained or bilious on insufficient grounds, particularly if they are convinced that their liver is out of order, and that what they need is "to get the bile off the stomach."

We now come to the second view of this phenomenon, viz., that the block in the bowel occurred comparatively late, after the descent of the bile past the occluded point. This is probably the correct explanation in the majority of cases in which the stools are described as meconium after due and sufficient observation.

It is generally accepted that the bile is *formed* by the third month, but one experiences great difficulty in finding any reliable observations proving at what date it *descends* to the bowel, and along the bowel. According to Savariaud, the bile is discharged into the duodenum in the fifth month, and during this and the sixth month it is confined to the small gut. By the seventh and eighth months it has reached the ascending and

transverse colons, and by the ninth month, the descending colon, sigmoid, and rectum. By these dates one would expect a constriction taking place in the duodenum in the fifth month, or in the ileum in the latter part of the sixth month, to cut off some of the bile in the bowel below the block. Cordes holds that the bile is discharged into the bowel by the fourth month, so that a duodenal block occurring even at that date would shut off meconium below.

*Obstruction by inspissated meconium alone* may be mentioned here, though, strictly speaking, not included in our subject. The writer remembers seeing a post-mortem examination on an infant who died apparently from congenital intestinal occlusion, without operation, and no definite obstruction was found, but only thick meconium in the small gut and tenacious mucus lower down.

Ecoffet records another such case, in which enterostomy was performed. There was no real block, but a gradual diminution of calibre, most marked at about the ileo-cæcal valve. The meconium was very thick, and "below the artificial anus the contents got as stiff as putty, and could not be moved on." The large gut contained "sebaceous matter" not coloured by bile.

Pearce Gould, in connection with his case, says, "The cæcum, the lower four inches of the ileum, and the first four inches of the colon were filled with a firm whitish plug of inspissated mucus, of the consistence of cheese, which was firmly applied, but not adherent, to the mucous membrane. In the colon, beyond this plug, were found several masses of milk-white, firm mucus. Below this, the colon and the rectum were empty and contracted to the size of a clay tobacco pipe stem." It was assumed that the plug was deposited by the third month of foetal life.

Burdick records a similar case. "The last 4 c.m. of the rectum were normal. The remainder of the large gut, and last part of the ileum, were patent to the probe, and filled with a yellowish cheesy material. As the ileum gradually expanded (when followed upwards), this was replaced by normal meco-

nium." The colon was the "size of a goose quill." This writer also quotes another case (Jennings'), "The upper part of intestine is filled with meconium. Farther down the contents become thicker, and ropy in consistence. The colour is lost, and they have a white cheesy appearance." Burdick considers that a case recorded by Hepburn, in which no definite obstruction was found, was of a similar nature to the above. In this case the ileum had an "undeveloped character until about the junction with the jejunum, where it passed insensibly, without any stricture, into healthy bowel distended with meconium." This was drained. "Two other children, previously born of the same mother, were the subjects of imperforate anus."

In one of Grisel's cases the meconium was too thick to escape through the enterostomy wound, and in a case recorded by Eustache there was obstruction caused by meconial mass in the descending colon.

These cases afford convincing proof that congenital obstruction may be caused wholly and only by the abnormally firm consistence of the meconium, apart from any stricture. They correspond to the "obstruction by foreign bodies" in the classification of acquired obstructions.

7. *The Peritoneum.*—In most cases this is normal, and shows no signs of any preceding or present peritonitis. When a short band intervenes between the blind ends of the gut (as in Cases 5, 6, and 7), the peritoneum is seldom quite so smooth and glistening over the band as over the normal gut, and it would probably be correct to say that there has been some strictly localised peritonitis, whatever be the cause of the obstruction, just at that point. In Case 8a (a youth of 19) there is more marked roughening over the stenosis, which favours the view that this obstruction is acquired. In Cases 20 and 24 we have marked and definite evidences of foetal peritonitis; the former is simply a conglomerate mass of adherent coils, and in the latter the peritonitic adhesions were so plentiful as to mask the relations of the pelvic viscera, block the ureters, and apparently also the colon. From the character of the adhesions, and from



their results (hydronephrosis, etc.), one can say definitely that they had been present probably several months, and certainly several weeks.

It is impossible to decide how much peritoneal disturbance found at birth should be required as a sufficient proof of a preceding peritonitis. Theremin paid particular attention as to whether the mesentery and omentum were shortened at all, or the glands enlarged, as well as to adhesions, and consequently he found evidence of peritonitis in a large proportion of cases.

However, taking as evidence of a preceding peritonitis only fairly obvious peritoneal changes, as is usually done, one finds that only about one-seventh of the cases have their presence noted, and in only a very small minority of the cases is the omentum or glands mentioned at all.

8. *Other Abnormalities.*—These are not very common. As might be expected, the most frequent is *imperforate anus* or rectum. This was present in Cases 14, 19, 21, and 24. In Case 23 the imperforation, though commencing in the descending colon, involved also the rectum. Besides these five, the writer has come across the records of (at least) eleven others, making sixteen in all. In some of these, other deformities also existed. In Markwald's case there were atresias of œsophagus, duodenum, and rectum. In Marrigue's case there existed an atresia of the œsophagus, three blocks in small gut, and imperforate rectum. In the case of Hess there were imperforations of duodenum, rectum, and urethra, and in that of Voison the small gut emerged in a median fistula, the large gut was absent, and there were also atresia ani, spina bifida, ectopia vesicæ, split pubis, and defect of penis.<sup>2</sup>

In Cases 9 and 11 the foramen ovale was found widely open. In Case 22 there was an abnormally large ductus arteriosus, and in Case 24 a double hydronephrosis, due to foetal peritonitis. Another abnormality, coming next to imperforate anus in interest (though not in frequency), is the persistence of a Meckel's diverticulum with a stricture. In Case 8 this occurs, though the diverticulum is quite minute. In Case 8a, which is only

doubtfully congenital, the diverticulum has become invaginated, and so caused obstruction. Others have also recorded the presence of a diverticulum, together with an occlusion.<sup>3</sup> Other deformities which have occurred are cystic kidneys,<sup>4</sup> absence of stomach,<sup>5</sup> pyloric stenosis, absence of appendix,<sup>6</sup> double bowel,<sup>7</sup> deformities of hands and feet, absence of great omentum, hernias, and congenital heart disease. These cases in all, excluding hernias, come to about the same number as those in which an imperforate anus or rectum is present (either with or without other deformities). Therefore, we may say that in *at least* a tenth of the cases of congenital intestinal constriction some other abnormality is present, and that in half of these (or one-twentieth of the whole) an imperforate anus or rectum is the deformity, or is present with other abnormalities. Ducros, in his thesis on this subject, says that in monsters with congenital occlusion, the latter is most often multiple. He enumerates several writers as quoting cases supporting this contention.<sup>8</sup>

#### IV.—ÆTIOLOGY.

Several views are held as to the causation of these defects. Perhaps the most popular is that of Bland Sutton, who eluci-

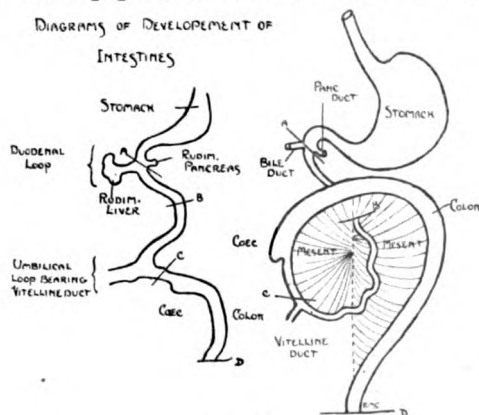


FIG. 11.

dated the subject in a short paper in 1889. Before that, Meckel had said that the bowel was developed in different

segments, and that any block was due to the failure of these segments to coalesce, and was always due to want of development of a part. Since Meckel's view as to the development of the bowel has not been generally accepted, his opinion as to the causation of these defects has also come to be disregarded.

One must here briefly refer to the orthodox view of intestinal development. Fig. 11 gives diagrams of the human alimentary tract. At about the fifth week this is only slightly curved, there being two loops with their convexities forwards, viz., an upper or duodenal loop, and a lower or vitelline loop, to the summit of which is attached the vitelline (or omphalo-mesenteric) duct, part of which persists sometimes (1 case in 50) as Meckel's diverticulum. More rarely still this may open at the umbilicus, forming a congenital fæcal fistula. Normally, a small part of this vitelline loop is present in the umbilical cord at the tenth week. Abnormally it persists there too long, and so may be cut with the cord at birth, or may be cut off more slowly by the closing in of the abdominal walls and tightening of the umbilical ring around it. In this case a length of bowel may be absent from the abdomen.

The duodenal loop develops into the duodenum, and from it are seen two outgrowths—an anterior, which forms the liver, and a posterior, which gives rise to the pancreas.

The upper limb of the vitelline loop forms most of the small intestine; the lower limb forms the rest of the small and most of the large bowel. At a later stage the lower arm of the vitelline loop rises over and crosses the upper, as shown in Fig. 10, and one can see how it comes about that the small bowel, in the neighbourhood of the duodeno-jejunal flexure, comes to perforate the base of the transverse meso-colon.

The most frequent points of congenital occlusion are marked in these diagrams. They are:—

- A. In the duodenum, near the biliary papilla.
- B. At the duodeno-jejunal flexure.
- C. In the lower ileum or near the ileo-cæcal valve; and most often of all, of course, in
- D. The rectum, with which we are not at present concerned.

The diverticulum which forms the liver begins in the fourth week, and the gland develops in the second month from the two halves of the now divided diverticulum. The common duct is then formed by a further pushing forth of the duodenal wall. The villi and Lieberkühn's glands form in the second and third months, but Brunner's glands not till the end of the fourth month. The vitelline duct is normally obliterated by the third month, the intra-abdominal portion disappearing first, perhaps because of its being stretched.

1. *Bland Sutton's View* is that occlusion of the alimentary tract always occurs at the site of an "embryological event." When the dimple which is to form the anus does not unite with the hind gut we get an imperforate rectum or anus. Similarly, but more rarely, the stomodeal invagination which forms the mouth does not unite with the fore gut, and we get an imperforate pharynx. Should the normal closure of the vitelline duct, instead of not being carried far enough (as when Meckel's diverticulum remains), be carried too far, so as to encroach upon the normal bowel wall at its point of attachment, a stricture or obliteration of the bowel in this situation comes about, and this may also result conceivably by simple traction on the bowel by the remains of the duct.

Occlusions of the duodenum are somewhat similarly explained by this view as being connected with the development of the liver and its ducts. It is not quite so simple here, however, as, of course, the bile duct is not normally obliterated as the vitelline duct is, and is not usually found obliterated, in these cases of duodenal occlusion. If a marked change in the attachment and development of the mesentery be held to be an "embryological event," this view can explain those occlusions which occur at the duodeno-jejunal flexure, where the small gut penetrates the root of the transverse meso-colon.

A congenital volvulus of Meckel's diverticulum can certainly cause an obstruction, as in Carwardine's case, where the twisted diverticulum formed a cyst, completely blocked the descent of meconium, and caused the bowel below to be typically "worm-

like." Kolliker is quoted by Taillens as saying that the vitelline duct normally gets axially rotated and that this rotation going too far may affect the gut.

Three specimens of ileum were shown at the Pathological Society by Hudson some years ago. The first had a short diverticulum, presumably Meckel's, and there was also a slight narrowing of the gut at the place of its attachment. In the second there was a membranous septum, in the same part of the bowel, perforated by a hole a quarter of an inch across. In the third there was an obvious stricture which had caused a fatal result in the same situation. Hudson also, in the account of these cases, tabulated the various grades of deformity in connection with the diverticulum, both those due to imperfect closure of the vitelline duct, such as umbilical fistula, cysts, etc., and those due in his opinion, to too complete closure of the duct, such as the strictures with which we are dealing. After reading his concise record it is almost forced upon one that developmental reasons are amply sufficient to account for the defect, in this situation, at any rate.

In support of this view the following points may be urged:—

(a) Undoubtedly in most cases the obstruction is at one of the points named, that is, either near the biliary papilla or in the lower ileum. In this connection it is of some importance to know the usual position and extreme limits of attachment of Meckel's diverticulum. This is commonly said to occur in the ileum between one and three feet above the cæcum. Lamb, in 1893, collected 185 cases. In only 98 of these was the situation definitely stated. The process was present double as often within one foot of the valve as in any other foot of bowel, and two-fifths of the cases occurred here. The last *two feet* of the ileum contained no less than three-fifths of the whole number. On the other hand several cases occurred high up in the ileum, in the jejunum, and even a few in the duodenum. These last were probably diverticula due to other causes. Generally speaking, therefore, it is true to say that the diverticulum may occur anywhere in the jejuno-ileum, but with increasing fre-

quency from above downwards, being exceedingly rare in the jejunum, and most common within a foot of the ileo-cæcal valve.


If we now turn to the cases of occlusion which occur in the jejuno-ileum, and consider, for clearness, only the single ones, we find the same thing holds, viz., they are very rare in jejunum, and increase in frequency right down to the valve. About half of all single occlusions in the jejuno-ileum are specifically stated to be in the lower ileum, and in many more the situation is not definitely stated.

In the writer's series, seven cases (besides 8a, which is doubtfully congenital) are in the lower ileum (5, 7, 8, 10, 11, 12, and 13), three of them being just above the cæcum (5, 7, and 12). In the duodenum the situation of the block does not lend so much support to Bland Sutton's view. The number of obstructions above the papilla is about equal to those below it, almost half of the latter being away from the papilla at the duodeno-jejunal flexure. A fewer number are said to be opposite the papilla. Generally speaking, the obstruction is in most cases *near* the biliary papilla, *i.e.*, not far above or below it, or else at the point where the small gut passes under the meso-colon. In some cases of new-born infants a kink has formed and caused obstruction in this last situation, and one can imagine that such a kink developing earlier might cause a constriction there.

In about one-fourth of all cases the blocks are either multiple, or, if, single, situated in the colon, and so can hardly be explained by Sutton's views.

(b) The fact that the obstruction is generally single favours this view as opposed to the inflammatory view.

(c) A persistent diverticulum is sometimes found co-existing with an occlusion. In Case 8 a minute diverticulum exists immediately below an occlusion. In Case 8a, also, the invaginated diverticulum is attached just below the stricture. In Pretty's case "a diverticulum of conical shape, equal in size to the tip of one's finger, was seen on the intestine about an inch before the latter gradually enlarged into the cul-de-sac"; here the



process was apparently attached *above* the stricture. Diverticula were also present in the cases of Carini, Lameris, and Cazin. In the two cases of the latter the diverticulum opened at the umbilicus, and the bowel was stenosed below. Clogg and Ahlfeld have also recorded the presence of this process, but probably in their cases the atresia of the gut was caused differently, being cut off by the closure of the umbilical ring (q.v.i.), which process is certainly favoured by the presence of a diverticulum. Craig found an obstruction apparently caused by an "omental tag" attached to the short band of the atresia. This has been held by some, probably wrongly, to be the remains of a diverticulum. It is hard to explain why the occlusion should so rarely in these cases be just at the point of attachment of the process; in nearly all it is either above or below.

If we hold Sutton's view, it is arguable that a persistent diverticulum (due to a defect of closure), and an occlusion (due to excess of closure) should never co-exist; but if we imagine that the obliteration takes place irregularly or that the tendency to obliteration spreads abnormally it might account for these and similar cases.

(d) Anal (or rectal) and pharyngeal congenital obstruction are, by common consent, regarded as accounted for by developmental defects; and, therefore, by analogy, one would be inclined to favour a similar view for the deformities with which we are dealing. Also, imperforate anus is the most frequent associated deformity, as mentioned above (in at least one case in 20). This rather indicates a common cause.

(e) Similar strictures are rarely seen in the aorta, at the point of attachment of the ductus arteriosus, or lower down, where the right aortic arch is attached in foetal life (Rolleston).

(f) In several instances there appears to be a family tendency to this or similar defects. Thus, the cases recorded by Forrer (in which there was a funnel-shaped diminution of calibre from lower jejunum down to rectum) was of the same parents as that of Franke (in which anastomosis was made for an ileal

block, with fatal result through a stitch giving way). Wünsche, in recording his case of a duodenal obstruction, says that a brother and two sisters died with the same symptoms. Craig notes that the mother of his case had had a previous child who died from some form of congenital obstruction, almost certainly not an imperforate anus. Carver found an exactly similar defect in each of twins.

Hepburn says, in regard to his case of obstruction (in which there was probably no definite anatomical block), "two other children, previously born of the same mother, were the subjects of imperforate anus." In Fairland's case of double bowel, one branch of which ended blindly, he tells us that the mother had had a child years before who died from the "absence of any outlet to the bowel," and that the parents had been in dread of a similar deformity.

(g) In most cases the peritoneum is normal, and shows no definite signs of any antecedent peritonitis.

(h) Anatomical considerations. In a case such as that of Buchanan, where the bile duct opened on the upper surface of a partial duodenal septum, and the pancreatic duct on the lower surface, it is hard to resist the view that the valve is a kind of exaggerated biliary papilla. Other cases are very similar.

Histological appearances of valvular occlusions certainly support some developmental view, though the same can hardly be urged where there is a band.

2. *Snaring of a loop of bowel at the navel* by the closure of the abdominal walls certainly accounts for some cases. As noted above, at the tenth week of development the apex of the vitelline loop is enclosed in the cord, and if it is retained there too long, the normal closure of the umbilical ring will cut it off. This is well illustrated by Fig. 12, which represents a case of obstruction in a new-born infant recently recorded by Mr. G. E. Waugh. In this case, as in others similar, there was a persistent diverticulum, which would keep the loop of bowel from being withdrawn into the abdomen abnormally. Mr. Waugh found a strangulation, but no occlusion, and the umbilical ring



had to be enlarged to permit of the intestine being returned. Six inches of bowel were excised, and the child lived a month after, and died apparently from other causes. One can easily understand, from this figure, that had the umbilical ring closed rather earlier the loop might have been cut off and even completely disappeared, as it would have had no blood supply.

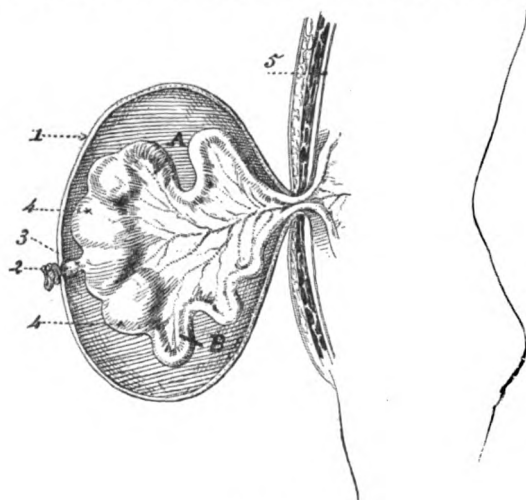


FIG. 12.

One of Mr. Clogg's cases was very similar to the above, the gut for functional purposes ending blindly in the hernial sac and a persistent diverticulum being present. Lameris records atresia of the lower ileum found in an umbilical hernial sac, a persistent duct here also being present. In Ahlfeld's case not only was the ileum but also the cæcum and part of the colon in the sac and cut off from the rest of the bowel. The vitelline duct was held responsible. Case 14, with a long segment of bowel missing, and others like it have almost certainly a similar causation.<sup>9</sup> The same may be said of the case of Moreau, where there were two narrowings in connection with an umbilical hernia.

3. *Epithelial Occlusions.*—Tandler cut serial sections of several (eleven) human embryos, and in his paper gives figures of them.

In these one sees that the duodenum at from 30 to 60 days is filled, or almost filled, with epithelial cells. Tandler says that at this stage the epithelial development gets ahead of the mesoblastic. He says the same is found to obtain in other tubes, *e.g.*, trachea and œsophagus, and he observed it also in the embryos of the guinea-pig and the rat. Forssuer confirms the above as regards the duodenum. Kreuter found that a great part of the intestinal canal of vertebrates loses its lumen in the early part of embryonic life, after it has already existed, and later recovers it again. He holds that congenital atresias are due to the non-occurrence of this normal canalization.

To support this view one would expect to find in sections of these occlusions some excess of epithelial elements, partly or completely closing the lumen.

The above three views of the ætiology of this condition may be truly called developmental, though the second approximated to the following, which hold that the obstruction is due to what may be termed a *fœtal accident*.

4. *Volvulus*. N.B.—Many cases of occlusion have been described, more particularly by Continental writers, as being due to “axial rotation.” On studying these one finds that this rotation is nearly invariably around the mesentery (*i.e.*, the axis of the loop) rather than around the long axis of the bowel. Therefore, these cases are considered here to be *volvuli*. It is hard to see how rotation in the long axis of the bowel can take place to any degree with an intact mesentery, though this appears to have occurred in Streubel's case (which is noted below under “kinking of bowel”).

*Volvulus* is probably a fairly frequent cause of the deformity, and is supported by such authorities as Braun and Bretschneider. *Congenital volvulus* has been observed by Pitt (where it was two feet above valve), Eschbach, and Cripps. In the latter case, the twist was also two feet above the valve, and the infant died of peritonitis. There was no meconium in the undilated part of gut, so presumably the torsion must have been present in some degree since the sixth month.

Bednar records two cases of volvulus shortly after birth, at 10 and 16 days respectively. In the first, the whole of the ileum and the ascending colon were affected, and the meso-colon was found to be extra long; the other is said to have involved all the small bowel, presumably excluding the duodenum. It is probable that in some early cases the twist affected the whole of the vitelline loop, from which are developed jejunum, ileum, and most of the colon (as far as the splenic flexure, or further). In the case of Thomas, the whole of the gut between the end of the duodenum to beyond the splenic flexure was absent. However, in this case there were other developmental defects present. In the cases of Epstein and Soyka and of Kleinwächter and Eppinger there were atresias of small intestine and of ascending colon, in each case the cause given being rotation round the mesentery. Very similar cases have been recorded by several others,<sup>10</sup> and particularly by Gärtner, who collected 13 cases in 1883.

In occlusion from this cause one would expect to find either a double narrowing, as when both limbs of a loop have been constricted, or a long gap, where the constriction has been severe enough to cut off the blood supply of the loop entirely and cause its disappearance. This view, therefore, explains cases of multiple atresias more easily than any developmental view can, particularly those where there is an even number of blocks. As, however, in many cases the exact number is not stated, one cannot say whether this theoretical consideration is confirmed by experience. In Emanuel's case, with five blocks, he explains the first as being due to a kink at the formation of the mesentery, and the others as being due to volvuli. Multiple cases attributed to this cause were also recorded by Fischer, Therman, and Kleinwächter and Eppinger (another case).

In the writer's series those cases which might *possibly* be due to this cause are:—

Case 14 (Mr. Steward), with a long fine connecting band.

Case 15, with five complete blocks.

Case 16, with three complete blocks.

Case 17, with one complete, one incomplete, and probably others.

Case 18, with double constriction of jejunum.

Case 19, with double constriction of ileum.

Of these, Case 14 is just as likely to be due to a strangulation at the navel, or to an earlier developmental defect, as there was an imperforate anus also. Cases 15 and 16 have an odd number of blocks, and, therefore, we must suppose, if due to volvuli, that one limb of an involved loop became far more constricted in the process than the other. Case 17 is quite as likely to be due to peritonitis. Cases 18 and 19 are more probably due to volvuli than to any other cause as yet advanced.

As against this view of causation, it seems to the writer that if a volvulus occurs in a fœtus, and the constriction at the neck of it becomes so tight as to destroy the bowel at the affected spot in one or both limbs of the involved loop, and possibly also damages the loop itself, one would hardly expect the twist to become disentangled spontaneously. Rather, we should expect it to become permanently fixed in the faulty position, and the loop involved perhaps to disappear. If this does take place, each gap represents where a loop of gut has disappeared. One can only say that the appearances make this improbable, as the connecting band is generally smooth, and there is no such puckering of the mesentery as one would expect.

5. *Kinking of Bowel*.—Cases attributed to this nearly all occur at the duodeno-jejunal junction. Thus, in one of Durante's cases the duodenum (or early part of jejunum) was found to be sharply bent over a mesenteric band, and so complete obstruction was caused. In another of his cases the angle at the duodeno-jejunal flexures "seemed to be too acute." In Streubel's case the ileum made a half turn on its long axis, and so caused kinking and obstruction. Emanuel attributes the first block in his case to a kink. In the case of Hess, the block was apparently caused by the "pressure of the meso-colon." In some of these cases there was no real occlusion or stenosis.

6. *Intussusception*.—The view that this is a frequent cause is due chiefly to an observation of Chiari, who in case of congenital atresia ( $4\frac{1}{2}$  c.m. long, and situated 15 c.m. above the ileo-cæcal valve) found in the lumen of the bowel below the stricture a cylindrical structure, visible with the naked-eye. This proved on examination to be a piece of intussuscepted gut.

Braun, in a case of occlusion in a similar situation, found a piece of necrotic gut one c.m. long quite free in the upper end of bowel below the block. This ensheathing bowel appeared on casual examination to contain only white matter. This structure is figured in Braun's paper, and there is no doubt as to its being the remains of bowel. It is of interest to note that in this case there were traces of a fibrous peritonitis.

Kirchner found a peculiar structure in the intestine beneath a jejunal occlusion, which was *possibly* the remains of a cast-off intussusception. He gives an elaborate description of the microscopical appearances, but nothing very definite can be made out. A case in which the history points to the passage of a sloughed intussusception per anum in a child of five days, with recovery, is recorded by Carter.

The above are the only observations supporting this view which can be found, but it appears to the writer that much may be urged in support of this way of regarding these cases ætiologically.

(a) The cases of Braun and Chiari definitely prove that the commonest class of obstructions (*i.e.*, where a short connecting band is present) *may* be caused by an invagination.

(b) Braun's case also proves that an intussusception may be missed unless carefully looked for.

(c) Both congenital occlusions and intussusceptions may occur anywhere, but are commonest in the ileo-cæcal region.

(d) The ileo-cæcal orifice is very small (absolutely and relatively) in infants, and, therefore, would be more likely to strangle and cut off small bowel invaginated through it. It may be urged against this that in the most frequent form of intussusception, the ileo-cæcal, forming 44 per cent., the valve forms the apex of the intussusception.

(e) In many cases altered blood has been passed per anum, and in others matter recorded as being meconium was probably only coloured with blood pigment and not bile. These phenomena are easily explained if we assume that the block was due to the above cause.

(f) It will agree with either single or multiple occlusions.

(g) The microscopic examination of these bands points to an acute hæmorrhagic inflammation of the mucosa, such as one might expect to accompany the sloughing off of an intussusception.

*Against this view* is the fact that the bowel below the obstruction, being small and contracted, would be expected to show if it contained a sloughed-off intussusception, and does not do so. Therefore, we must suppose that the intussusception becomes partially or completely absorbed. Also we should expect the mesentery to be folded or pleated at the place of the obstruction, and this is not so. It has been argued that the intussusception found in the above cases was the *result*, and not the cause, of the occlusion.

7. *Strangulation through a Mesenteric Orifice*.—Blot quotes a case where a congenitally obstructed bowel passed twice through such an orifice, but it is quite possible that the obstruction was independent of the orifice. Deville quotes a case where the coils of small gut were found to be lodged between the layers of the mesocolon, so that on opening the abdomen only the colons could be seen till the transverse mesocolon was split.

In a case of Rutherford's, of obstruction a few days after birth, a loop of lower ileum was found strangled and partly sloughing through a mesenteric orifice. Rutherford remarks that such holes in the mesentery are probably of congenital origin, tend to occur in the same place, have smooth edges, and may be circumscribed by vessels. There may be a thinning without an actual hole. In none of these cases was an obstruction certainly caused by such a strangulation.

Having now considered the above "foetal accidents" as causes of congenital occlusion, we come to another view which has received very wide support, viz., the

8. *Inflammatory View.*—This supposes a preceding inflammation, either an enteritis or more often a peritonitis. It was strongly supported by Theremin who, in 1877, collected practically all the then recorded cases, paying special attention to the state of the peritoneum and retro-peritoneal glands.

The existence of foetal peritonitis as a pathological entity was proved by Simpson in 1838. He collected 23 cases, of which he saw nine himself, in two years. The majority of the infants were still-born, and several more only just survived birth. The peritonitis was not due apparently to appendicitis. The exudate was mostly fibrinous, but more rarely puriform. Three of the mothers were certainly, and another three possibly, syphilitics. Two infants had hepatitis, and others enlarged glands and spleen. Simpson concluded that there is "sufficient evidence for establishing the pathological fact that the foetus in utero is occasionally the subject of peritoneal inflammation." What is of most interest, however, in the paper is the fact that two of the above twenty-three infants had a congenital stenosis of the bowel. They were recorded by Billard (lower end of duodenum), and Morgagni (multiple atresias in ileum and colon).

In the writer's series of cases, No. 24 shows marked signs of a preceding pelvic peritonitis obstructing the colon and ureters; and Nos. 17 and 20 also show similar but less definite signs. Druitt's case is very like these, the bowel constituting a tangled mass with many partial and complete occlusions.

As to the *cause* of this peritonitis, it is very indefinite. Probably it is often specific, as indicated in the figures given by Simpson above. But of the 328 or more so far recorded, there does not seem to be an abnormally large proportion of infants known to be syphilitic. Savariaud supposes that the toxic element is transmitted from the mother by the rupture of small placental vessels. Mauclaire, in a case with many occlusions and definite signs of an old fibrous peritonitis, found tubercle bacilli in the gut wall and in the mesenteric glands. Our information of the bacteriology of this condition is extremely scanty, so that no one can be dogmatic as to the cause.

In MacCallum's case he found a tough band across the lower ileum, almost amputating the gut, the calibre of which was diminished by adhesions in other places. Waterston records that about one inch above the ileo-caecal valve a peritonitic band compressed the gut, caused it to get dilated, and appeared as if in time it would have severed it. Weber found the duodenum fixed to the portal fissure of the liver with a strong old peritonitic band. Definite signs of an old preceding peritonitis have been recorded by many others.<sup>11</sup> In most of these cases the obstruction was in the lower ileum or duodenum if single, or there were multiple occlusions. These situations correspond roughly to those of obstructions supposed to be due to developmental defects, and it may be supposed either that the obstructions were independent of, or, perhaps, even the cause of, the peritonitis, or that these regions, the ileo-caecal particularly, were specially prone to inflame. As stated above, foetal appendicitis has not yet been proved.

In considering this subject, one must remember how frequently adhesions are absorbed. The result of operations depending for their success upon the persistence of adhesions (*e.g.*, ventro-fixation, etc.), is notoriously uncertain. In infants they are absorbed more readily than in adults, and in the foetus probably more quickly still. Therefore, there may be very few signs at birth, even after an extensive foetal peritonitis, but the writer considers that it is pushing this view too far to say that a congenital stricture may be the *only* evidence of a preceding peritonitis, as does Fieldler.

It is obvious that other modes of production of this defect (snaring at navel, volvulus, intussusception, etc.) must be accompanied by some slight adhesive aseptic peritonitis during the strangulation process, and any slight peritoneal changes found later may be looked upon as causative, when, in reality, they are the effect. Very rarely one end of the constricted or divided bowel has been found open. This is either due to a perforation of the distended bowel or to a constricting agent cutting into gut, or to a very acute inflammation (Poelmann),<sup>12</sup> These cases would, of course, leave signs of peritonitis.



The chief argument in support of this view, as opposed to that of Bland Sutton, is that the latter cannot explain multiple occlusions or those away from the situation of an "embryological event." The inflammatory view is supported by a large number of writers on the subject (Theremin, Silbermann, Fieldler, etc.), but in the opinion of the writer it has held too prominent a place. Only about one-seventh of the cases show signs of peritonitis, and in the greater part of these it must have occurred later than the obstruction. It must be noted that as the bile descends in the small gut at about the fifth or sixth month any obstruction must have taken place earlier if there is no bile below it (as is generally the case). In the case of an infant born at term, therefore, any adhesions found must have been present three months or more if they are sufficient to account for the occlusion.

9. *Vascular Anomalies* as a cause of occlusion. Jaboulay injected the mesenteric vessels, and found that the superior mesenteric artery did not send the usual branches towards the ileo-cæcal region, where the stenosis was. Küttner only found a single vascular arcade opposite an atresia in place of the four or five normal arcs. Wyss, in two cases of duodenal atresia, found an absence of the pancreatic duodenal artery. The vascular deficiencies are themselves traced to intra-uterine infections by Durante and Tailens. A congenital absence of ascending and transverse colon was traced to a vascular atrophy by Nowicki. It is possible that the bowel may be closed by *pressure* of a vessel. Von Haberer figures such a case in an adult, the superior mesenteric artery in the root of the mesentery compressing the end of the duodenum.

Grisel, in a case of obstruction with many adhesions, found a generalised endarteritis, but in this case there was no definite anatomical block in the bowel. The view that this defect may be due to *embolism* is attributed to Virchow. There is no evidence that *thrombosis* ever is a cause of this anomaly.

10. *Pressure of head of pancreas* has been supposed to cause a duodenal obstruction in the cases of Mohrmann, Heyman, Serr,

Voron, and Mickulicz. Though the head of the pancreas intimately embraced the stenosed or obstructed part in these cases, one cannot allow that it was, therefore, necessarily causative.

11. *Inflammation and Ulceration* of the intestinal mucosa.—This view is supported by Kirohner, Thorel, Markwald, and others. Certainly there was ulceration of the bowel found in these cases, but it is probable that it was not causative, but was a consequence. In the case of Markwald there were also atresias of the cesophagus and rectum, which would rather incline one to adopt a developmental view of the duodenal obstruction also. The microscopic examination of connecting bands, however, as stated above, strongly supports the view that the origin of the lesion is some condition of the mucosa resulting in exfoliation of its epithelium and accompanied by hæmorrhage.

If an early inflammation or ulceration of the mucosa *were* the cause of the obstruction, one would hardly expect to find any ulceration at birth, as the stricture is formed during the healing process.

12. *Hypertrophy of the Valvulæ Conniventes*.—This has been held to be the cause of some of the valvular obstructions. Against it may be urged the microscopical appearances as recorded above. Also, it must be remembered that the valvules are not well developed at birth. Grawitz, in recording a case of double obstruction in the ileum, both diaphragmatic, one partial and the other complete, says that a fold of mucous membrane may grow together round the bowel and so obstruct.

13. *Neoplasm*.—Bagourd found a "papillary adenoma" in the duodenum just above the stricture in his case, but it apparently was not causative. Wiederhofer found the terminal loop of ileum adherent to an alveolar carcinoma of the liver in an infant of three days!

14. *Other rare conditions* which have been advanced as causative are: Compression of bowel by a mesenteric cyst (Henning), or by a cyst in the iliac fossa (Schott); traction by Hernia, or by omental tag (Craig, q.v.s.), and, finally, spasm. Gaukler and Nau showed three cases in infants at the Société Ana-

tomique de Paris in which there were false narrowings, microscopically seen to be due to spasm. The bowel had "l'aspect d'un chapelet."

*To sum up the aetiology* of this condition, it is certain that no one view can explain all cases. The writer places the causes thus in order of frequency—

1. Developmental defects, including snaring at the navel.
2. Foetal accidents, *i.e.*, intussusceptions, volvuli, kinks, etc.
3. Foetal diseases, *i.e.*, peritonitis, ulceration, etc.

#### V.—SYMPTOMS.

The symptoms and signs are those usual in intestinal obstruction, and need not detain us long.

*Vomiting* is practically always present, and usually appears on the second day. At first the ingested milk is returned, but soon the vomit becomes green, and sometimes, but rarely, it is described as faecal, as in Case 22. Vomiting of blood is often recorded,<sup>13</sup> or of blackish matter, almost certainly altered blood,<sup>14</sup> as in Cases 5 and 15. Those cases in which there was vomiting of bilious matter, even when the obstruction was high in the duodenum, above the biliary papilla, have been alluded to above.

The only account of a complete occlusion which the writer has come across, *without* any vomiting, was that of Helmholtz (multiple atresias of the jejuno-ileum) where the child died when two days old only, perhaps from other causes, as it had a congenital heart and cirrhosis of the liver. In Hey Groves's case of a tight stenosis of the ileum in a child who lived to twenty months there was no vomiting. Neither was there in Burdick's case, but there the obstruction was only caused by inspissated mucus.

*Constipation* is usually absolute, though a little colourless or greyish mucus may be passed spontaneously. If an injection be given it will be found that only one or two ounces of fluid can be inserted, and that all it brings away is a little mucus. Those cases in which the obstruction is above the entrance of the bile

duct usually pass scanty green motions, as might be expected. Bilious matter has often been passed, spontaneously, or with an injection, in other cases also (q.v.s. page 175).

The passage of blood or black matter per anum has often been noticed, mostly in the same cases in which there was hæmatemesis.

*Distension* is present in some degree in nearly all cases. It is not there at birth usually, though in the case of MacCallum it was so extreme as to hinder parturition. In this case the intestinal obstruction was due to a foetal peritonitis, which had caused many marked adhesions amongst the coils of bowel. Rarely the distension is localized.

*Icterus* of a slight degree is present at birth in some cases, but may disappear later. It is supposed to be due to "hypertension in the biliary tree" (Savariaud). It is hard to say whether it is present in a larger proportion of these cases than in normal new-born infants, but in the latter it almost always develops a few days after birth.

*Visible peristalsis* is sometimes, but rarely, observed, proceeding from left to right in a duodenal occlusion, and more irregularly if the obstruction is lower.


The *subtegumentary collateral circulation* is, rarely, visible, and probably more often in those cases due to a preceding peritonitis.

*Anuria* is present in some cases, apart from those in which the ureters are obstructed (e.g., Lobligeois).

*Convulsions* have rarely been recorded.

## VI.—DIAGNOSIS.

A full consideration of the above symptoms, taking care not to attach too much weight to the passage of any motion which may be altered blood, or to the passage of a small amount of greenish motion, will, in the great majority of cases, enable a diagnosis of obstruction to be made with ease. The point of difficulty is to determine the site of the obstruction, and to do this one must first eliminate the presence of any ano-rectal imperforation.



To this end, attention must be paid to the following points:—

(a) *Inspection of the anal region.*—If there is no anal depression one need not, of course, investigate further, but, short of this, the anus may appear too small, or too far back, or the ischia may be too close together.

(b) *Palpation with the little finger.*—An ano-rectal block is almost always within two inches of the anus, and the dilated bowel above can be felt to bulge when the child cries.

(c) *Exploration with a Sound.*—In some cases where the finger appears obstructed a sound can be made to pass two inches or further. If it only passes two or three inches there is almost certainly *not* an ano-rectal obstruction. The writer would suggest that a small size of gum elastic bougie be first used, and if this passes, say, three inches, larger sizes be tried. In this way one could probably soon insert the little finger for a sufficient length to make the diagnosis more certain. In Case 4, which was the only one of the series thoroughly examined and diagnosed by the writer during life, after the passage of a catheter per anum and the injection of soap and water, one could pass the finger much more easily.

(d) *Enemata.*—The small amount of fluid which one can inject has already been mentioned. If nothing comes away, after repeated enemata, but almost colourless mucus, a diagnosis of intestinal obstruction, most probably due to a congenital occlusion, may be made.

Too much attention should not be paid to a *history* of an obstruction felt in the rectum till a thorough examination has been made as above. In Cases 12 and 15 the notes say that the rectum seemed to be obstructed two inches above the anus, and also in Cases 9 and 22, and in several others, the rectum was thought to be obstructed when really the block was much higher, the resistance felt being due to the contracted bowel. Many surgeons have incised perineally, and lost time through an insufficient anal examination.

When an imperforate anus or rectum is also present, as in 14, 19, 21, 23, and 24, it is impossible to diagnose a second obstruc-

tion above it previous to operation. In Case 14 Mr. Steward could feel through the imperforate rectum a rounded swelling which bulged downwards when the child cried. This was doubtless the dilated blind end of the ileum, but it was separated from the anus by some feet of impervious bowel.

If, in such a case, one has great difficulty in finding, or cannot find, the dilated cul-de-sac of the rectum, it should give rise to a strong suspicion that there is a higher occlusion, and an abdominal section should be proceeded with at once. The same step should be taken if a perineal incision only discovers a contracted coil of bowel, or a coil which, when opened, does not liberate meconium.

If by the above means an ano-rectal block has been excluded, one may attempt, often fruitlessly, to diagnose more exactly the site of the occlusion. Duodenal obstructions give symptoms approaching to those of pyloric stenosis, *i.e.*, more marked vomiting, less marked distension (than in lower occlusions), and peristalsis, if visible, from left to right. The symptoms of a duodenal occlusion above the biliary papilla would be exactly similar to, though more severe than, those of a pyloric stenosis. The latter, however, very rarely gives symptoms till the second or third week of life, so that there would not be much practical difficulty in distinguishing between the two.

If the stomach were demonstrated to be enlarged, either by percussion, by skiagraphy, or by lavage, of course that would be strongly in favour of a duodenal obstruction, as opposed to one lower down, but would not distinguish a duodenal from a pyloric occlusion, unless it could be made out from a skiagram, which is doubtful. The value of this skiagraphic method in these cases has yet to be demonstrated. It is doubtful, especially in the case of low obstructions, whether the bismuth would descend low enough or soon enough to be of service. It will probably prove of great value in cases of stenosis merely.

The appearance of meconial stools, or of bilious matter in the vomit, may throw light on the point as to whether the obstruction is complete, or as to whether (if it is complete) it is situated

above or below the entrance of the bile duct. As stated above, there have been many cases in which meconium stools have been recorded in blocks below the duct, and other cases where bilious vomiting has been recorded in blocks above the entrance of the duct. Therefore, too much stress should not be laid on these points, but the supposed bilious matter should be tested for bile pigment, and examined microscopically for lanugo hairs before deciding that it is really bilious matter.

By careful consideration to the above points, one should be able to decide certainly that the case is one of congenital obstruction of the bowel above the rectum, though the possibility of general peritonitis should be considered.

#### VII.—PROGNOSIS.

The prognosis is absolutely bad in cases of complete occlusion. Here, apart from operation, of course, life cannot be maintained, though Case 1b, where a child lived nine months with an apparently complete duodenal septum, appears to be an exception to this rule, as does the following extraordinary case. Von Murat presented an observation at the Académie Royale de Médecine de Paris in 1823 of a beggar man, aged 70 years, who lived in a village near Nancy, who had paraplegia, and in whom there was an imperforation of the anus and of the urinary passages also. The whole of the food residues were said to be vomited, and one cannot discover what became of the urinary excretions. At the same meeting of the Académie, M. Cloquet said he possessed observations on similar cases, and referred to an earlier paper, which, however, cannot be traced by the writer. Baux (quoted by Esmarch) tells of a girl aged 14 years with a complete rectal occlusion, who vomited her fæces every two or three days. These clinical curiosities, however, in no way relieve the prognostic gloom of a case of complete congenital occlusion.

A well-marked stenosis may cause little or no symptoms, as in the cases of Hudson, Silcock, and Moore; and a stricture which is quite tight may be compatible with several years of life, as in Case 8a, in another of Hudson's cases, in one of Perry and Shaw's cases, and in that of Carling.

At least 82 cases of operation for this condition have now been recorded, and not one has recovered. Nevertheless, the writer considers that in a small minority of cases in the future there should be some chance.

#### VIII.—TREATMENT.

In the Appendix will be found a list of 82 cases of operation. There is no practical utility in going into the details of these, suffice it to say that the great majority were enterostomies. In Case 15 Mr. Steward succeeded in stitching the contracted bowel to the anal margin by the perineal route, after dealing with an imperforate anus, but this would appear to be a very difficult procedure. More often a perineal incision has been found quite useless, and has had to be followed by a laparotomy.<sup>15</sup> Braun, having diagnosed a high rectal stricture, incised from the anus in the first instance, but had to proceed to make a left iliac, then a right iliac, and finally a median abdominal incision!

As regards the enterostomies, the bowel has been drained at all points, of course, several times as high up as the duodenum. In most cases the infants have died within a day or two of the operation, as in Cases 4, 5, 7, 11, 14, 16, 18, and 22. A small minority seem to have much more resisting power, as Case 9, which was operated upon at two days old, and lived till twelve days. Tischendorf's case lived till three weeks (fifteen days after operation). Case 2, which was not operated upon, also lived till three weeks.

*Anastomoses.*—At least nine of these have been previously recorded for this condition, and the writer adds three more, viz., Cases 1 and 7 and an unrecorded case of Arbuthnot Lane. Here a gastro-enterostomy was performed for a duodenal obstruction and an attempt made to squeeze on the gastric contents through the contracted bowel below. In Case 1 also a gastro-enterostomy was performed, and a third was made by Mohrmann, in which case the child lived two days after.

Five anastomoses have been made connecting small bowel to small bowel by Oliver (who did the end-to-end operation),



Braun, Bretschneider, Leloir and Grosse, and Franke. All these died shortly after except the patient of Franke, which is said to be the only one in which such an anastomosis has functionated. The infant lived three days, and then died from general peritonitis consequent upon a stitch giving way.

Four anastomoses connecting small and large gut are on record: Case 7, upon which Mr. Moynihan operated; Mangoldt's case, with five points of occlusion; that of Wanitshek; and, finally, that of Simmonds. This last case is of interest in that a second occlusion was found post-mortem below the site of the anastomosis.

In some of the above cases it was definitely stated that at the autopsy it was found that the meconium had not penetrated into the worm-like bowel below the obstruction.

In the face of the above facts, it will be a matter of opinion whether or not any operative procedures should be taken. Probably, if the unfortunate infant happens to be born in an institution or in connection with some great maternity charity, it will be submitted to operation, whilst if it is not thus situated it will be allowed to die untampered with.

The writer considers that as soon as the diagnosis is made, laparotomy should be performed, firstly, because the condition cannot be diagnosed with absolute certainty from others occurring at, or soon after, birth, such as volvulus, intussusception, internal strangulation, etc., which may be remedied; and, secondly, because he believes that in the future some of these cases will be saved. There is no reason, for instance, why a block in the duodenum above the papilla should be fatal if gastro-enterostomy be performed soon enough, nor why a colon obstruction, or even one near the valve, should *invariably* prove fatal.

The non-success of enterostomy has led Clogg and others to advocate the abandonment of this operation in these cases. The opinion of the writer is that this is not a scientific position to adopt. It is certain that enterostomy, *as a final operation*, has proved, and is likely to prove, useless. Infants apparently suffer

more from the exclusion of a part of the alimentary tract than do adults. But it is equally useless to attempt a short circuiting operation in a toxæmic and collapsed infant. Therefore, in cases where the obstruction has lasted long, the only immediate hope is drainage. If the child dies within two days, as the majority do, it is certain that it would not have survived with a more severe operation. If it shows some rallying power, as a minority do (*e.g.*, Case 5), an anastomosis may be made as soon as its condition permits. Most certainly if the case be diagnosed early an anastomotic operation should be done, and enterostomy is never needed. But in practice one finds that, because of the exceeding rarity of the affection, it is often not diagnosed till the infant is in a bad condition.

Clogg suggests that the worm-like bowel should be distended at the operation by water pressure. The writer considers this to be a suggestion the value of which cannot be exaggerated, as it is certainly useless to join up a dilated loop to one which is so contracted as not to allow anything to pass. This dilatation was performed post-mortem by the writer in Case 4, and also by Polaillon in his case. In Case 4 the contracted bowel after this dilatation appeared almost normal. A nozzle connected to a tap may be used, or even a large syringe, but considerable pressure is needed, and massage of the bowel at the same time helps. The success of the future depends upon how much of this worm-like bowel it is found possible to dilate up without killing the infant from shock. This procedure will also show the absence of another atresia below—a most important point, as in some cases (*e.g.*, Simmonds), a second block has been overlooked at the operation.

The treatment of *incomplete occlusions* is comparatively simple. Probably a lateral anastomosis without excision of the stenosed part of bowel would be the best line to adopt in most cases.

Perhaps a few dogmatic rules for the treatment of these cases may be excused:—

1. Satisfy yourself that there is no definite obstruction within two inches of the anus.

2. Use spinal anaesthesia if possible (as in the case of Waugh, page 187), and take all precautions against shock.

3. Perform a median laparotomy.

4. If there be a complete and single block in the lower ileum or colon, dilate up all the contracted bowel below it by water pressure, and do an anastomosis in the quickest possible way. If the block be higher up, take the same line.

5. With a duodenal obstruction above the papilla, do a gastro-enterostomy.

6. With a duodenal obstruction below the papilla: (a) if a stenosis merely (so that the bile can pass downwards), a duodeno-enterostomy may be attempted, or, failing that, a gastro-enterostomy; (b) if a complete atresia, with all the gut below contracted, close the abdomen.

7. If multiple atresias exist: (a) close together, they may be excluded, and the dilatation and short circuiting proceeded with as above; (b) widely separated, close the abdomen.

8. If the infant be in a very bad condition, do an enterostomy and see if it rallies.

9. In operating for imperforate anus, if the dilated bowel above cannot be fairly easily found, or if, when found, it does not contain meconium, or seems contracted, open the abdomen.

#### IX.—SUMMARY.

Congenital intestinal occlusion is generally single, and most often occurs in the lower ileum. The next most frequent site is in the duodenum, near the biliary papilla. Multiple occlusions most often occur in the jejuno-ileum.

As regards aetiology, the occlusions are due to many causes, but most frequently to developmental defects, particularly in connection with the vitelline duct and with the development of the liver. Other causes are accidents to the foetal bowel, such as volvuli, intussusceptions, and kinks; or foetal diseases such as peritonitis and intestinal ulceration.

The symptoms are those of obstruction, and the diagnosis is made by excluding any ano-rectal imperforation. The prognosis is absolutely bad, and the treatment advocated is early laparotomy followed by dilatation of the contracted gut by water pressure and lateral anastomosis.

*Leicester, 1912.*

#### APPENDIX A.

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Notes of some Cases of Stenosis which have survived for three months or longer.

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Schwytzer records that in an infant three months old, dead of inanition, there was found, besides a pyloric stenosis, a narrowing of the colon thought to be congenital.

In Buchanan's case, a child of 18 months, there was a septum at the level of the biliary papilla, with a central aperture only 2·5 mm. in diameter. The symptoms (vomiting and convulsions) had only recently appeared, and the child vomited so rarely whilst in hospital as to lead to an error in diagnosis.

Hey Groves tells of an infant who remained quite well whilst it was breast-fed (one year), but after weaning it lost flesh and became distended; exaggerated peristalsis could also be seen, but there was no vomiting. It died at 20 months, and there was found a hard, unyielding stricture, hardly admitting a probe, in the ileum. A large quantity of vegetable debris had collected above the stricture.

Hartman, at the Société de Chirurgie, showed a skiagram of a girl of five years, in which a great narrowing of the pelvic colon was seen. There had been constipation from birth, and sometimes there was no evacuation for six weeks.

Hudson showed at the Pathological Society, in 1889, a portion of ileum from a boy of eight years, so strictured as to hardly admit a probe. There was a history of old abdominal illness. This specimen can be seen in the Middlesex Hospital Museum, and is almost certainly congenital in origin.

In Case 8a, as recorded above, the patient, aged 19 years, had a constriction of ileum possibly congenital in origin.

In Silcock's case a partial congenital septum was found in the duodenum of a woman of 34, who died from an epithelioma of the ascending colon. This septum bulged downwards in the duodenum like the

finger of a glove, and the obstruction was not enough to cause any dilatation of the duodenum above it. The specimen is in the Museum of St. Mary's Hospital.

Okinczyc quotes the case of an adult female, in which he found a "segmentary atrophic atresia" in two places in the descending colon, with dilatation between. It is definitely stated not to be due to spasm. He says he has seen another somewhat similar case, in which there was a history of constipation.

Perry and Shaw shortly describe preparation 2,428 in the Royal College of Surgeons' Museum. Here, near the pylorus, the duodenum is reduced to a very small diameter by contraction with slight thickening of its walls. There is no history, but the specimen appears to be from an adult. It is doubtful, however, whether it is congenital in origin or not.

Moore has recorded, in a man of 40 years, who died of bronchitis, the presence of an apparently congenital stricture at the duodeno-jejunal flexure due to a ring of mucous membrane.

Carling records another possibly congenital stricture at this point (duodeno-jejunal flexure) in a woman aged 47 years. There was no microscopical evidence of a cicatrix at the stricture, which, when fresh, would only admit a probe. Presumably it could not have been that size from birth, and symptoms had only existed for one year.

Hudson showed at the Pathological Society, the same day as his other case, a diaphragmatic stricture of the ileum in a man of 62 years who had showed no signs nor symptoms of it. This specimen is also in the Middlesex Hospital Museum.

Finally, Keith mentions that specimen 2,522a in the Royal College of Surgeons' Museum is a stricture at the ileo-cæcal junction in a woman of 62. Syphilis is mentioned as causative, but Keith thinks the stricture is very probably congenital.

## APPENDIX B.—LIST OF OPERATIONS.

### 1. ANASTOMOTIC OPERATIONS.

<i>a. Gastro-enterostomies.</i>				<i>c. Small bowel to colon.</i>			
Case 1	...	...	1911	Case 7	...	...	1906
Mohrmann	...	...	1905	Simmonds	...	...	1900
Arbuthnot Lane (circ.)	...	...	1905	Mangoldt	...	...	1896
				Wanitschek	...	...	1894
<i>b. Small bowel to small bowel.</i>							
Leloirier and Grosse	...	...	1905				
Braun	...	...	1902				
Oliver (end to end)	...	...	1901				
Bretschneider	...	...	1898				
Franke	...	...	1898				

Making a total of twelve.

2. OTHER OPERATIONS, ENTEROSTOMIES, ETC.

Case 11	...	...	1910	Chaput	...	...	...	1894
" 4	...	...	1905	Prochownick	...	...	...	1891
" 14	...	...	1904	Aly	...	...	...	1891
" 14a	...	...	1904	Fischer	...	...	...	1891
" 5	...	...	1900	Bland Sutton	...	...	...	1889
" 9	...	...	1900	Lyot	...	...	...	1888
" 6	}	Undated.		Tischendorf	...	...	...	1887
" 16				Atkin	...	...	...	1885
" 18				Kermisson	...	...	...	1884
" 22				Lucke (in Gartner)	...	...	...	1883
Burdick	...	...	1908	Pearce Gould	...	...	...	1882
Bergallonne	...	...	1907	Roser (in Schottelius)	...	...	...	1881
Davis and Richardson	...	...	1907	Fairland	...	...	...	1879
Grisel (three cases)	...	...	1905	Davies-Colley	...	...	...	1878
Clogg (two cases)	...	...	1904	Polailon	...	...	...	1876
Souter	...	...	1904	Dehio	...	...	...	1872
Pery	...	...	1903	Créde (in Ahlfeld)	...	...	...	1873
Letoux	...	...	1903	Jacobi	...	...	...	1869
Kuliga	...	...	1903	Valenta	...	...	...	1864
Hepburn	...	...	1902	Marjolin (in Cazin)	...	...	...	1862
Salva Mercadé	...	...	1902	Malgaigne (in Laborde)	...	...	...	1861
Lilienfeld	...	...	1902	Druitt	...	...	...	1860
Simmonds	...	...	1900	Verneuil	...	...	...	1858
Narath (in Laméris)	...	...	1900	Depaul	...	...	...	1856
Martens	...	...	1900	Burgrave (in Poelman)	...	...	...	1855
Broca and Savariaud (in Ecoffet)	...	...	1900	Nelaton (in Depaul)	...	...	...	1855
Wyss	...	...	1900	Depaul	...	...	...	1855
Mauclaire	...	...	1900	Pretty	...	...	...	1854
Sick	...	...	1900	Voillemier (in Savariaud)	...	...	...	1846
Villemin	...	...	1899	Voison	...	...	...	1804
Clarke	...	...	1899	Making a total of 70, including a few cases such as Case 15a, and that of Pearce Gould, where there was no definite occlusion.				
Gidionsen	...	...	1898					
Hess	...	...	1897					
Carwardine	...	...	1897					
Mangoldt	...	...	1896	With the dozen anastomotic operations above, the grand total comes to 82.				
Anders (in Hecker)	...	...	1896					
Dalziel	...	...	1895					
Good	...	...	1894					

## APPENDIX C.—LIST OF CASES.

## 1. DUODENUM.

*Above biliary papilla—*

Case 1a (Keith, and Roe and Shaw)

Anderson (1889)  
Aubery  
Champneys  
Collum  
Cordes  
Crosby Leonard  
Emerson  
Guyot  
Hempel  
Hervey  
Hirschsprung  
Hobson (1893)  
Jackson  
Levy (q.v. Hirschsprung)  
Markwald  
Michel  
Northrup  
Perry and Shaw  
Porak and Bernheim  
Rosenkranz  
Sick  
Silbermann  
Taillens  
Theremin (two)  
Voron  
Wilks  
Wyss

*Opposite papilla—*

Cases 1 and 2

Aly  
Buchanan  
Dohrn  
Durante  
Hammer  
Hecker (1856)  
Mohrmann  
Serr  
Silcock  
Simmonds  
Theremin (three)  
Wallmann  
Weber (q.v. Kuliga)

*Below papilla—*

Billard  
Born  
Brindean  
Commandeur  
Crooks  
Eastes  
Hess  
Heyman  
Jacobi  
Kermisson  
Pery  
Rokitanski  
Schuller (q.v. Hirschsprung)  
Shaw, L. K.  
Sick  
Speyer  
Theremin (two)  
Trumpf  
Wünsche  
Wyss

*At duodeno-jejunal flexure—*

Case 3

Baron  
Carling  
Cohen  
Commandeur  
Darier  
Durante  
Gallico  
Grisel  
Hasselmann (q.v. Hirschsprung)  
Klein (q.v. Meckel)  
La Baume  
Moore  
Pied (q.v. Meckel)  
Willet

*Unclassified--*

Case 1b (Keith).

Albers  
Bacon (q.v. Cohen)  
Ferber  
Freeman  
Schukowski  
Schutz  
Seibert  
Sonden  
Lane (unrecorded)

2. JEJUNO-ILEUM (unclassified).

- |                               |                                |
|-------------------------------|--------------------------------|
| Cases 6, 10 and 14            | Bednar                         |
| Albers                        | Bretschneider                  |
| Archambault                   | Braun                          |
| Broussolle                    | Brodelet                       |
| Cases                         | Broca                          |
| Davies and Richardson         | Carver                         |
| Dehio                         | Carwardine                     |
| Durante                       | Cazin                          |
| Ducros                        | Charrier                       |
| Forster                       | Chiari                         |
| Gartner                       | Clarke                         |
| Gibert (two)                  | Clogg                          |
| Good (q.v. Kuliga)            | Dalziel                        |
| Grisel                        | Davies-Colley                  |
| Groves, Hey                   | Depaul (two)                   |
| Hecker (1896)                 | Desgranges                     |
| Helmholz                      | Dohlhoff (two)                 |
| Henock                        | Forrer (q.v. Kuliga)           |
| Hoffmann                      | Franke                         |
| Hubbard                       | Gaupp                          |
| Hufeland                      | Gidionsen (q.v. Bretschneider) |
| Huttenbrenner                 | Grisel                         |
| Jungnickel                    | Guersant                       |
| Keith (fig. 10)               | Henrich                        |
| Kirchner                      | Heyfelder                      |
| Küttner (1872)                | Hobson (1885)                  |
| Lyot                          | Horch (q.v. Meckel)            |
| Nobiling                      | Hudson (two)                   |
| Pilliet                       | Jacoby                         |
| Polailon                      | Laméris                        |
| Rolleston                     | Lassus                         |
| Rose                          | Letoux                         |
| Schellong (q.v. Kuliga)       | Litré (q.v. Ducros)            |
| Schlegel (q.v. Braun)         | Lobligeois                     |
| Sick (two)                    | Lucae                          |
| Streubel                      | MacCallum                      |
| Sutton, Bland (two)           | Mangoldt                       |
| Theremin (three)              | Martens                        |
| Thevenet                      | Nancy (q.v. Hirschsprung)      |
| Thompson (q.v. Kuliga)        | Nelaton                        |
| Thore                         | Oliver                         |
| Tobeitz                       | Osiander                       |
| Valenta                       | Petit                          |
| Villemin                      | Pretty                         |
| Waterston                     | Serand (q.v. Ducros)           |
| Wiederhofer (q.v. Silbermann) | Souter                         |
| Wilkinson and Knox            | Southey (q.v. Ducros)          |
| Willet (Lockwood's Case)      | Sutton, Bland                  |
|                               | Theremin                       |
|                               | Tischendorf                    |
|                               | Valenta                        |
|                               | Wanitshek                      |
|                               | Weber (q.v. Kuliga)            |
|                               | Weiland                        |
|                               | Weill and Pehu                 |
|                               | Wiederhofer (1859)             |
- Lower ileum, and in the immediate neighbourhood of the ileo-cæcal valve—*
- Cases 5, 7, 8, 8a, 10, 11, 12 and 13
- Andrews  
Atkin



## 3. COLON.

Cases 22, 23 and 24—  
 Anderson (1891)  
 Baudelocque (q.v. Meckel)  
 Bednar 2580  
 Benninger (q.v. Ducros)  
 Fyfe  
 Grossman  
 Grüneberg  
 Hartman

Kristellar  
 Loseke (q.v. Meckel)  
 Meckel  
 Mukhanoff  
 Nowicki  
 Okinczyc  
 Schwytzer  
 Smith  
 Solger

## 4. MULTIPLE OCCLUSIONS.

Cases 15, 16, 17, 18, 19, 20  
 and 21  
 Adie  
 Albers  
 Bergallonne  
 Boyd  
 Büttner (q.v. Meckel)  
 Cazin  
 Calder (q.v. Ducros)  
 Curtius (q.v. Meckel)  
 Demme  
 Dohrn  
 Druitt  
 Durante and Siron  
 Emanuel  
 Epstein and Soyka  
 Fieldler  
 Fischer  
 Fliessburg  
 Gilbert  
 Grawitz  
 Grisel (two)  
 Guersant  
 Hecker (1857)  
 Henoeh  
 Helmholz  
 Hirschsprung  
 Kleinwachter and Eppinger (two)

Kronlein  
 Kuliga  
 Küttner (1846)  
 Laborde  
 Lucke (q.v. Braun)  
 Mangoldt  
 Marrigues (q.v. Meckel)  
 Maucisire  
 Minich  
 Morgagni (q.v. Kuliga)  
 Poelmann  
 Preisich  
 Righetti  
 Roederer (q.v. Meckel)  
 Schafer  
 Schott  
 Schottelius  
 Schnizlein  
 Schuller (1855)  
 Schuppel  
 Simmonds  
 Steinthal  
 Theremin (two)  
 Thorel  
 Turner  
 Verneuil  
 Voillemier (q.v. Savariaud)  
 Ziegler

## 5. UNCLASSIFIED.

Case 21  
 Ahlfeld  
 Behm  
 Bernheim  
 Blot  
 Bonnaire (two)  
 Cleeman  
 Clogg  
 Clopat (q.v. Ducros)  
 Craig  
 Daniel (q.v. Meckel)  
 Dodd  
 Fairland  
 Farabeuf

Heister (q.v. Ducros)  
 King  
 Lataret  
 Leloir and Grosse  
 Lilienfeld  
 Moreau (q.v. Ducros)  
 Neilson  
 Otto  
 Prochownich  
 Robinson  
 Sale  
 Thomas  
 Voison

*Cases of obstruction due to thick meconium—*

Burdick  
 Ecoffet  
 Eustache

Gould, Pearce  
 Hepburn  
 Jennings (q.v. Burdick)

APPENDIX D.—REFERENCES.

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2. Other cases—Baudeloque, Dehio, Fischer, Grossman, Klein, Schuppel, Thomas, Villemin.
3. Ahlfeld, Corini, Cazin, Clogg, Lameris, Pretty.
4. Gärtner, Lücke.
5. Roederer, q.v. Meckel.
6. Grisel, Osiander.
7. Fairland.
8. Büttner, Calder, Hensinger, Hufeland, Osiander, Roederer.
9. Hecker (1896), Lilienfeld, Schellong.
10. Dohrn, Durante, Good, Latarjet.
11. Dohrn, Grisel, Helmholz, Martens, Nancy, Sick, Thompson, etc.
12. Others—Grisel, Helmholz, Schlegel.
13. Grisel, Durante (in two cases), Emerson, Levy, Seibert, Serr, Wyss.
14. Darier, Ferber, Freeman, Hervey, Porak, and Bernheim.
15. Anders, Atkin, Fairland, Hecker (St. Petersburg), Letoux, Lucke, Pretty.

N.B.—Those references marked with an asterisk have not been accessible to the writer for verification.

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# GALL-STONES. COLIC DUE TO BLOOD CLOT.

A RARE COMPLICATION FOLLOWING REMOVAL  
OF STONES FROM THE COMMON BILE DUCT.

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By

R. P. ROWLANDS.

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As a rule, patients do remarkably well, and complications are exceptional after the removal of gall-stones. The greatest fear is that hemorrhage may take place when jaundice is deep and has been continuous for a long time. Even this risk can be lessened by the injection of an alien serum before operation and by carefully tying all bleeding points during the operation. It is better still to avoid the risk altogether by operating early enough. The following case is so exceptional as to merit publication.

The patient, a stout man, aged 50, had many attacks of biliary colic which have been associated with jaundice on four recent occasions. The pain has sometimes lasted as long as two days, and has been very severe in the upper part of the right side, extending through to the right shoulder. When I first saw him at Out-Patients three weeks before operation he was recovering from one of these attacks, and the jaundice was subsiding. He was tender above and to the right of the umbilicus over the common bile duct. I therefore concluded that he had a stone in the common bile duct, and I strongly advised an operation. While waiting for admission he had several more attacks of colic associated with temporary jaundice.



*Operation.*—A Kocher's incision was made, a few adhesions were separated, and the gall-bladder was seen to be contracted. Two large stones were felt in the common bile duct. An incision was made directly over them after Morison's pouch had been well packed. The stones were barrel-shaped, faceted, and nearly one inch in diameter. A finger was passed up into the hepatic duct and downwards along the common duct without finding any more stones. There was a little oozing of blood from the upper part of the incision in the common duct; therefore, a single catgut suture was used to stop the bleeding and to close this part of the large opening. The packs were removed, and a large tube containing a wick of gauze was placed in Morison's pouch, and the abdomen was closed in layers. The operation lasted twenty minutes, and the patient did very well for three days, a good deal of slightly blood-stained bile coming away. Then he was seized with colic just like the old attacks. On shifting the tube and changing the gauze wick, the bile, which had ceased to run, escaped freely, and the pain subsided. Several similar attacks developed during the next few days, the patient feeling quite well in the intervals. The tube was removed on the sixth day, and very little bile escaped after this time. On the tenth day he had an unusually severe attack, shivered, and became more deeply jaundiced, and his strength began to fail. It was, therefore, decided to re-open the wound without delay. On doing this a small collection of dark blood was found below the liver in Morison's pouch, and a large swelling was felt in the position of the common bile duct. The old incision, which had healed, was re-opened, and about two handfuls of clot escaped. The blood shot out, and was evidently under great pressure. The last portions to come away were white and organising. The finger was passed into the common duct to explore, and it was remarkable to find that the duct had immediately shrunk nearly to its natural size. A small squirting artery was seen and tied at the upper end of the incision in the duct. When the passage was proved to be clear, a tube was passed into the duct, and the wound was drained. An injection of horse

serum was given. The operation afforded immediate relief, and the patient made a good recovery. The case illustrates the great importance of—

1. Taking great pains to stop all oozing during operations for jaundice.
2. Providing free drainage from the common bile duct in all cases.
3. Recognising that recurrence of colic after the careful removal of all stones may be due to blood clot.



# METABOLISM DURING RECTAL FEEDING.

WITH A NOTE ON THE PRESENCE OF CREATIN  
IN THE URINE OF CHILDREN.

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By

N. MUTCH, M.A., M.B., B.C. (Beane Research Student in  
Materia Medica),

AND

J. H. RYFFEL, M.A., B.C., B.Sc.

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Food materials cannot with reason be introduced into the rectum to alleviate starvation until the possibility of their absorption and metabolism through this channel has been demonstrated. Recently the subject has been investigated by Langdon Brown. He criticised the statement of earlier observers, that as much as 50 per cent. of the protein injected may be absorbed, as being based on the analysis of material obtained by washing out the colon, a process notoriously difficult to complete. He concluded from comparison of the effect, on the quantity of urinary nitrogen, of protein given per rectum with that of equal amounts given by the mouth, that protein, even when partly pre-digested, cannot be absorbed from the colon in sufficient quantity to warrant the inconvenience of its administration. He also concluded that the absorption of starch and fat was negligible.

Laidlaw and Ryffel examined the fæces and urine of a man who for 47 days was fed rectally with a mixture of egg-white, starch, and peptonised milk. The fæces, which were acid owing to the presence of lactic acid, with traces of butyric acid, con-

tained a large quantity of unaltered food. The urine corresponded both qualitatively and quantitatively with that of continued starvation, containing only 4.64 grm. of nitrogen per diem. In this case it is clear that the materials employed were of little value. The production of acids, presumably by bacterial action, adequately explains the intolerance of the large intestine observed during the experiment.

The following experiment was designed to test the validity of the contention that various foodstuffs introduced into the rectum disappear from its lumen after a moderate interval.

Case 1.—D. S., female, aged 6 years:—

TABLE I.

8 a.m. to 8 a.m. Day of Expt.	10 a.m.	4 p.m.	10 p.m.	4 a.m.	8 a.m.
1	Purgative given in the evening			Breakfast 6 a.m.	
2	Mouth feeding ceased	Chloroform administered 8 p.m., and operation performed for closure of cleft palate		225 c.c. dextrose solution given per rectum	Colon washed out with 1 litre of normal saline
3	225 c.c. normal saline per rectum	225 c.c. dextrose solution per rectum	225 c.c. normal saline per rectum	225 c.c. dextrose solution per rectum	Colon washed out 1 litre saline
4	225 c.c. saline	225 c.c. dextrose	225 c.c. dextrose	225 c.c. saline	Do.
5	200 c.c. peptonised milk per rectum	225 c.c. saline	100 c.c. peptonised milk	225 c.c. saline	Do.
6	200 c.c. peptonised milk	225 c.c. saline	200 c.c. peptonised milk	225 c.c. saline	Do.
7	200 c.c. dextrose	225 c.c. saline	200 c.c. dextrose	225 c.c. saline	Do.
8	200 c.c. dextrose	225 c.c. saline	200 c.c. dextrose	225 c.c. saline	Do.
9	Mouth feeding commenced	—	—	—	—

The whole of each enema was retained, and the tube was, in each case, washed through with water before withdrawal. The colon was washed out each morning very slowly through a tube and funnel immediately after the bladder had been emptied. None of the fluid escaped between the tube and the anal margin. The dextrose used contained mere traces of l  vulose, giving a much fainter resorcin reaction than did a specimen of Kahlbaum's pure dextrose. The peptonised milk was prepared according to instructions given in the Guy's Hospital Pharmacop  ia. The term, however, is a misnomer, for analysis showed the milk to consist chiefly of water, salts, fat, lactose, and altered caseinogen. Only traces of primary and secondary albumoses were present.

TABLE II.

## 1. NITROGEN.

Day of Experiment.	Nitrogen in Feeds.	Nitrogen Recovered.	Nitrogen Absorbed.
5	1.30 grm.	0.12 grm.	} 88 per cent.
6	1.50	0.09	
7	0.00	0.02	
8	0.00	0.11	
Total ...	2.80	.34	

The colon was washed out on three occasions after the last administration of milk, namely, after intervals of 10, 34, and 58 hours respectively, so that all traces of unabsorbed food must have been removed, together with an appreciable amount of intestinal secretion. The analyses show that only 12 per cent. of the nitrogen introduced into the large bowel could be recovered from the washouts, thus affording reasonable grounds for the conclusion that considerable absorption of protein can be effected by the colon.

TABLE III.

## 2. DEXTROSE.

Day of Experiment.	Dextrose in Feeds.	Dextrose Recovered.	Dextrose Absorbed.
7	38.4 grm.	0.07 grm.	} 99 per cent.
8	34.2	0.34	
Total ...	72.6	0.41	

As these results show, only 1 per cent. of the dextrose introduced into the rectum could be recovered from the washouts. Clearly then both dextrose and the nitrogenous constituents of peptonised milk can be absorbed from the large intestine either unaltered or after modification by the action of enzymes or of bacteria.

Dextrose is useful to the starving body in two ways—

1. Its oxidation is a source of energy, which spares the fat and protein of the tissues.
2. By supplying carbohydrate, the reserve of which is soon exhausted in starvation, it prevents the disturbances inseparable from a carbohydrate-free metabolism.

The former function is inadequately discharged. 170 grammes of dextrose is a large quantity to give per rectum daily, and this only yields 700 Calories, whilst an adult at rest requires about 2,000 Calories. It should, however, be easy to give dextrose in sufficient amount for the fulfilment of its second function, and if it fails to prevent metabolic disturbance, either it is not absorbed from the colon, or it is not metabolised in the same manner as when taken by the mouth.

The abnormalities in urine during starvation which are due to lack of carbohydrate are:—

1. The presence of acetone, diacetic acid, and oxybutyric acid, with the resulting acidosis and increase in the proportion of nitrogen as ammonia.
2. The presence of creatin.

Cathcart investigated the urine of a professional starving man. The total nitrogen fell continuously from 16 grm. per diem before the fast to 7·8 grm. on the fourteenth day. The ammonia ratio, which was 3·5 per cent. before the fast, rose to 14 per cent. on the tenth day. The creatinin fell continuously. Creatin appeared on the first day and continued throughout the fast, the amount being fairly constant (0·8 to 1·1 grm.) from the second to the fourteenth, or last day. Acetone and diacetic acid were present throughout. After the fast of fourteen days

starch and cream were given for three days, with the result that the nitrogen fell to 2.8 grm.; the ammonia ratio was slightly reduced, while creatin, acetone, and diacetic acid disappeared.

Cathcart has also shown that creatin which is present after a fast of forty hours disappears at once on an adequate diet of carbohydrate alone, but increases when fat or fat and protein are taken. Mendel and Rose have shown the same effect on creatin in rabbits.

In the following investigation an attempt has been made to employ these facts as tests of the metabolic utility of dextrose given by the rectum. So as to increase the quantity of dextrose, a 10 per cent. solution was used instead of 6 per cent., which is isotonic with blood. The methods of estimation employed were:—

Total nitrogen ... ..	Kjeldahl.
Ammonia ... ..	formalin and Shaffer.
Creatin and creatinin ... ..	Folin.
Acidity ... ..	Folin.
Acetone and oxybutyric acid ...	Shaffer.
Lactic acid ... ..	Ryffel.
Dextrose ... ..	polarimeter.

The first two experiments were performed on children, who differ from adults in that they feel the lack of carbohydrate much more acutely; acetone bodies appear sooner and in larger quantity and the proportion of creatin is increased beyond normal limits. (See note at end of paper.)

Case 1.—D. S., female, aged 6 years.

First day, purgative.

Second day, operation for cleft palate; chloroform.

Third and fourth days, dextrose per rectum.

Fifth and sixth days, peptonised milk per rectum.

Seventh and eighth days, dextrose per rectum.

Ninth day, food given by mouth.

Further details of the feeding are given in Table I., and the results obtained from the urine in Table IV.



TABLE IV.

8 a.m. to 8 a.m. Day of Expt.	Volume Urine in c.c.	Specific Gravity.	Total Nitrogen. Per cent.	Gm. per diem.	Proportion of Nitrogen as Ammonia.	*Total Creatinin. Per cent.	Gm. per di-m.	Ratio Creatin to Creatinin.	Acetone.	Diacetic Acid.	Indican.	Remarks.
4	—	—	1.54	—	15.8	0.119	—	1.77	+	+	0	40 gm. dextrose per diem. per rectum
5	215	1026	1.67	3.59	17.6	0.150	0.332	1.54	+	+	+	Peptonised milk 300 c.c. per rectum
6	545	1020	1.09	5.96	17.3	0.087	0.475	1.72	+	+	+	Peptonised milk 400 c.c. per rectum
7	725	1014	0.682	4.94	—	0.060	0.432	1.75	+	+	0	38.4 gm. dextrose per rectum
8	—	—	—	—	—	—	—	—	—	—	—	34.2 gm. dextrose per rectum
9	407	1007	0.676	2.75	16.4	0.088	0.358	0.88	+	+	0	Fed by mouth
10	415	1010	1.027	4.26	—	0.097	0.40	0.5	0	0	0	Do.
11	—	—	—	—	—	—	—	—	—	—	—	Do.
12	342	1018	1.048	3.58	—	0.070	0.241	0.11	0	0	0	Do.
13	510	1023	1.253	6.39	—	0.083	0.422	0.476	0	0	0	Do.

Day 6. Acetone, per cent. 0.099, per diem 0.537 gm.; Oxybutyric acid, per cent. 0.361, per diem 1.91 gm.—ratio to nitrogen  $\frac{1.91}{0.361} = 5.29$   
 Day 7. " " 0.078, " 0.566 " " 1.94 " "  $\frac{1.94}{0.566} = 3.43$

Lactic acid. Day 5, per cent. 0.019, per diem 0.0415 gm. Day 7, per cent. 0.050, per diem 0.305 gm.

\* By total creatinin is meant creatin plus creatinin, expressed as creatinin.

Variations in the nature of the enemata given do not appear to have produced any immediate corresponding variation in the excretion of acetone bodies, creatin, total nitrogen, or ammonia.

The presence of indican on the days when milk was given indicates bacterial decomposition of the protein of the milk. The lactic acid in the urine of a normal adult amounts to about 0.040 grm. per diem. In children it is less, so that the lactic acid was above normal on both the days mentioned, but especially on the seventh day of the experiment, when dextrose was given after milk. This can be explained by the conversion of some dextrose into lactic acid by bacterial action before absorption.

Case 2.—W. B., male, aged 3½ years.

First day, enema saponis; breakfast; operation for cleft palate; chloroform.

Second, third, and fourth days, 300 c.c. 10 per cent. dextrose and 300 c.c. normal saline per rectum per diem.

Fifth, sixth, seventh, and eighth days, 600 c.c. 10 per cent. dextrose per rectum per diem.

Ninth and following days, food given by the mouth.

The results of the urinary analyses are given in Table V.

The dextrose solutions were not retained so well as the salines, being partly returned in from three to four hours. On the sixth day the lactic acid in the urine was distinctly above normal (sixteenth day), a fact which suggests the formation of this acid in the bowel. The increased allowance of dextrose from the fifth to the eighth day reduced the loss of nitrogen considerably. The creatin also fell, but the ammonia continued to rise, and the acetone bodies were apparently unaffected. On the resumption of feeding by the mouth the creatin was higher for three days and then fell, but the acetone bodies showed a rapid immediate diminution. There was probably less carbohydrate given at first by the mouth than had been given per rectum. This would account for the rise of the creatin, but the rapid fall of acetone bodies is inconsistent.

TABLE V.

Day of Experiment.	Volume of Urine in c.c.	Specific Gravity.	Total Nitrogen.		Proportion of Nitrogen as Ammonia.	Total Creatinin.		Ratio Creatin to Creatinin.	Creatin Per diem.	Acetone.	Diacetic Acid.	Remarks.
			Per cent.	Gram. per diem.		Per cent.	Gram. per diem.					
2	204	1031	1.685	4.95	6.02	0.123	0.362	0.73	0.153	+	Slight	30 grm. dextrose per rectum
3	197	1030	1.92	3.78	13.1	0.176	0.347	2.8	0.256	+	+	Do.
4	210	1030	2.14	4.5	15.7	0.176	0.37	2.72	0.270	+	+	Do.
5	64	1027	—	—	—	0.107	0.068	2.56	0.049	+	+	60 grm. dextrose per rectum
6	145	1025	1.45	2.1	19.3	0.123	0.178	1.6	0.110	+	+	Do.
7	114	1026	1.66	2.49	25.4	0.162	0.185	1.54	0.112	+	+	Do.
8	108	1021	1.64	1.77	23.6	0.164	0.177	1.46	0.104	+	+	Do.
9	190	1019	—	—	—	0.142	0.27	1.54	0.164	+	Slight	Fed by mouth
10	225	1018	—	—	—	0.107	0.24	1.4	0.132	0	0	Do.
11	270	1018	—	—	—	0.094	0.254	1.17	0.137	0	0	Do.
12	390	1014	—	—	—	0.050	0.20	0.94	0.096	0	0	Do.
13 & 14	240	1019	—	—	—	0.079	0.19	0.89	0.089	0	0	Do.
15	—	—	—	—	—	0.025	—	0.77	—	0	0	Do.
16	250	1017	—	—	7.6	0.048	0.12	0.53	0.041	0	0	Do.
17	—	—	—	—	—	0.049	—	0.32	—	0	0	Do.

Before experiment, creatinin total per cent., 0.11; per diem 0.33 grm.

Ratio  $\frac{\text{creatin}}{\text{creatinin}}$  0.87

On 34th day, at home " " 0.1

Ratio 0.95

Lactic acid. Day 6th, per cent., 0.031; per diem 0.045 grm.

" 16th, " 0.004; " 0.010 "

Case 3.—G. J., male, aged 31 years.—Obstruction of the cardiac end of the stomach, probably malignant; general nutrition good. The exact duration of starvation prior to the administration of dextrose per rectum is not known, but it is certain that no food entered the stomach during the six days preceding the experiment.

Details of the feeding and the urinary analyses are given in Table VI.

The urine passed before the administration of dextrose would have been useful for comparison, but was lost. The effect of the anæsthetic on the third day in causing acidosis is very clear. That this acidosis was not due to the acetone bodies and lactic acid alone is also evident.

The dextrose given was completely absorbed. None was returned, and the washout at the end of the fourth day was neutral and contained neither dextrose nor lactic acid. Further, there was no abnormal excretion of lactic acid in the urine, so that there is, in this case, no evidence of the formation of lactic acid in the gut. Such formation, however, is not excluded, as the acid may have been completely absorbed from the gut, and its absorption must be rapid to cause an increased excretion in the urine.

Acetone was not determined, as thymol, which interferes with its estimation by Shaffer's method, was used for preserving the urine. Qualitatively dextrose had no effect on the acetone, but the amount present was very small, much smaller than that usually present in starvation. Oxybutyric acid, which was also present in very small quantity, was distinctly reduced by the administration of dextrose. When the first and second days are compared, the dextrose is seen to have caused a slight diminution in the ammonia ratio, which was, moreover, only slightly above the normal. After the second day there was a rise in the ammonia ratio due, however, to the anæsthetic. The creatin excretion showed a distinct progressive diminution under the dextrose, but the total nitrogen, which was high throughout, showed no such diminution.

TABLE VI.

Day of Exp.	Volume of Urine in c.c.	Specific Gravity.	Total Nitrogen. Per cent.	Grm. Per diem.	Percentage of Nitrogen as Ammonia.	Total Creatinin. Per cent.	Grm. Per diem.	Ratio Creatinin. to Creatinin.	Creatin. gm. per diem.	Acetone	Dia-cetic Acid.	Acidity c.c. $\frac{N}{10}$ per diem.	Remarks.
1	?	—	2.17	—	5.9	0.235	—	0.351	—	+	0	—	114 gm. dextrose per rectum
2	350	1036	2.84	9.95	5.31	0.368	1.29	0.405	0.371	+	0	292	170 gm. " " Energy 700 cal.
3	655	1025	1.37	8.97	8.47	0.170	1.11	0.382	0.308	+	0	419	Dextrose as previous day. Operation 2 p.m. Gastrostomy. Chloroform anæsthetic
4	475	1029	2.01	9.56	10	0.245	1.16	0.317	0.279	+	0	358	Dextrose as before
5	730	1031	2.39	17.45	8.53	0.330	2.41	0.533	0.934	Trace	0	505	Fed by Stomach Carbo-
6	550	1029	2.88	15.8	5.65	0.290	1.59	0.25	0.318	Minute trace	0	173	hydrate. Fat. Protein N. 31 gm. 6.1 gm. 0.95 gm. 37 7.4 1.2
7	425	1030	2.81	11.96	5.54	0.360	1.53	0.44	0.469	0	0	207	49 9.8 16
8	410	1033	2.90	11.91	6.9	0.360	1.48	0.27	0.314	0	0	325	98 19.7 3.1
9	365	1028	—	—	—	0.436	1.59	0.29	0.357	0	0	—	110 22 3.6
10	300 ?	1032	—	—	—	0.397	1.19 ?	0.13	—	0	0	—	240 47 8
17	1040	1028	—	—	—	0.123	1.28	0	0	0	0	—	210 110 25

Oxybutyric acid. Day 1, 0.017 per cent.—ratio to nitrogen  $\frac{1}{174}$   
 Day 2, 0.020 " " 0.070 per diem—ratio to nitrogen  $\frac{1}{172}$   
 Day 4, 0.013 " " 0.061 " "  $\frac{1}{178}$   
 Day 5, 0.0067 " " 0.049 " "  $\frac{1}{317}$

Lactic acid ... Day 4, 0.0085 " " 0.040 " "  
 Day 5, 0.0067 " " 0.049 " "

Small quantity of bile pigment present throughout, except day 17. Urobilin present from day 5 on. Indican only on day 17.

TABLE VII.

Day of Exp.	Volume of Urine in c.c.	Specific Gravity.	Total Nitrogen. Per cent.	Gm. per diem.	Percentage of Nitrogen as Ammonia.	Total Creatinin. Per cent.	Gm. per diem.	Ratio Creatin to Creatinin.	Creatin gm. per diem.	Acidity in c.c. $\frac{N}{10}$ per diem.	* Acetone R. S.	Remarks.
2	—	1021	1.77	—	3.83	0.169	—	0.71	—	—	+	Rectal saline 1140 c.c. None returned. Water by mouth
3	1840	1014	0.98	18.1	4.66	0.084	1.55	0.55	0.55	459	+	Do. do.
4	895	1019	1.21	10.9	7.5	0.133	1.19	0.57	0.43	455	+	111 gm. dextrose as 10% solution. Some returned.
5	445	1022	1.46	6.51	6.83	0.193	0.86	0.45	0.27	208	+	Water 568 c.c. daily Dextrose and water as before. 40 gm. dextrose only retained
6	745	1024	1.78	13.2	6.3	0.213	1.59	0.53	0.55	298	+	55 gm. dextrose as 10% solution by mouth.
7	520	1026	1.87	9.71	5.36	0.242	1.26	0.33	0.31	178	0	570 c.c. rectal saline
8	655	1022	1.57	10.3	4.86	0.188	1.23	0.33	0.31	172	0	Do. do.
9	795	1022	1.42	11.26	3.92	0.151	1.20	0.31	0.28	256	0	Milk and eggs by mouth.
10	1185	1021	1.44	17.03	—	0.111	1.31	0.31	0.31	—	0	Carbohydrate. Fat. Protein N. 46 gm. 40 gm. 10 gm.
11	1460	1018	1.30	18.9	—	0.080	1.17	0.31	0.28	—	0	57 80 13 70 100 15 80 180 18

The rectal salines and dextrose solutions were given four times daily, the colon being washed out once a day. On the 4th day some dextrose was returned, about a third of that given being lost in this way. On the 5th day two of the dextrose solutions were retained and two were returned in 1½ hours and 2½ hours respectively. The volumes were 400 c.c. and 700 c.c., and the amounts of dextrose 6.54 per cent. and 5.33 per cent. In the second case more dextrose was returned (37 gm.) than had been given in the previous feed (38 gm.). The first was alkaline and contained 0.03 per cent. lactic acid, the second slightly acid and contained 0.08 per cent. lactic acid. The wash out of this (the 5th) day amounted to 1260 c.c., and contained 0.65 per cent. sugar and 0.01 per cent. lactic acid; it was faintly alkaline. All the liquids returned from the rectum contained altered blood. Much blood was in this way discharged on the 3rd and 4th days, but only a small quantity on the 5th day. Indican was present in the urine on the 3rd day and on the 11th day only.

\* Acetone R. represents Rothera's test. Acetone S. the ordinary nitroprusside, caustic soda, acetic acid test.

On the fifth day feeding by the stomach was commenced with an artificial milk free from creatin and creatinin. Much less carbohydrate was given than had been given per rectum. This resulted in a very marked rise in the excretion of nitrogen, which was out of all proportion to the nitrogen in the food, and in another marked rise in the creatin excretion. The creatin excretion continued greater than that under rectal dextrose, even when 110 grms. of carbohydrate were given by the stomach. The acetone bodies, however, behaved quite differently, as they showed an immediate diminution when feeding by the stomach was commenced, in spite of the fact that far less carbohydrate was supplied. No conclusion can be drawn from the ammonia determinations owing to the disturbing effect of the anæsthetic.

This case, therefore, shows clearly that dextrose, when absorbed per rectum, is practically as efficient as carbohydrate taken normally in diminishing nitrogenous waste and creatin excretion, but is far less efficient in diminishing the acetone bodies.

Case 4.—W. B., male, aged 40 years.—Hæmatemesis. Details of the feeding and the urinary analyses are given in Table VII.

The results in this case are complicated by the fact that considerable bleeding took place from the stomach during the experiment. The bleeding was at a maximum on the third day, as shown by the rectal material, which was very rich in blood, the presence of indican in the urine, and the high urinary nitrogen. Under rectal dextrose on the fourth and fifth days the bleeding was much diminished, but it appears to have recommenced when dextrose was given by the mouth on the sixth day, and to have continued to the end, as shown by the presence of blood in the fæces and by the high urinary nitrogens.

Under these circumstances the apparent effect of rectal dextrose in restraining nitrogenous waste on the fifth day is exaggerated. As far as can be estimated the dextrose absorbed from the rectum on the fourth and fifth days together was about equal to that taken by the mouth on the sixth and seventh days. As in the previous cases, the rectal dextrose had an appreciable effect in reducing the urinary creatin, being apparently as efficient for

this purpose as dextrose by the mouth, but its effect in reducing the acetone bodies, and also apparently the ammonia ratio and acid in the urine, was considerably less than that of a similar quantity of dextrose when taken by the mouth.

Some evidence was afforded by the contents of the rectum of the formation of lactic acid from dextrose, but this did not take place to any great extent.

In those cases in which dextrose can be absorbed by the large bowel and subsequently metabolised, a very efficient means of supplying water to the body presents itself. The usefulness of isotonic salt solution (.9 per cent. sodium chloride) is undoubted; it has, however, the disadvantage that the substance used to render it isotonic is absorbed but not metabolised, so that unless excreted again it accumulates in the tissues. The body loses water by respiration, by sweating, and in the urine. That lost by respiration contains no salt, sweat contains only a small quantity (normally about .3 per cent., although the concentration may sometimes be higher), so that most of the chloride must be excreted by the kidneys. This service the kidneys can render only to a limited extent. The most concentrated urines rarely contain chloride equivalent to 2 per cent. sodium chloride, and the conditions under which rectal salines are required are unfavourable to excretion of chloride, even when the kidneys are normal. When these organs are abnormal their power of excretion is further reduced, and chloride readily accumulates in the body. The difficulty experienced even by normal people in excreting large quantities of salt is illustrated by the well-known fact that sea-water, which is approximately a solution of sodium chloride three and a half times as strong as isotonic saline, increases thirst instead of relieving it.

For this reason, in absence of a better substitute, it is desirable to use a saline solution which is as hypotonic as possible, such as that in general use, which contains about 0.7 per cent. instead of 0.9 per cent. sodium chloride. A 6 per cent. solution



of dextrose is also isotonic, and when absorbed is superior to saline in that the dextrose is oxidised to carbon dioxide and water. The 5 per cent. dextrose in saline, however, which has sometimes been used, is clearly undesirable, both because it is hypertonic and, therefore, irritating, and because it contains the same large quantity of salt as ordinary saline.

#### CONCLUSIONS.

The nitrogenous constituents of peptonised milk can be partly absorbed by the colon, but undergo some bacterial decomposition in the process.

Six to ten per cent. solutions of dextrose in water given by the rectum are, if absorbed, a more efficient source of water than is saline, especially when the kidneys are diseased. There is, however, considerable individual variation in the tolerance exhibited by the rectum towards these solutions.

Energy equivalent to about 700 Calories can in this way be supplied to an adult, with the result that the breakdown of the tissue proteins is considerably diminished, and the urinary nitrogen therefore reduced.

The effect of dextrose absorbed from the colon in reducing the output of creatin in the urine corresponds roughly to that of carbohydrate taken by the mouth. On the other hand, dextrose by the colon is definitely less efficient than carbohydrate by the mouth in inhibiting the excretion of the acetone bodies and probably also in reducing the acidosis of starvation.

This difference suggests that dextrose when absorbed by the colon is, to some extent, metabolised in an unusual manner. The only conceivable factor is the action of bacteria in the colon. That this plays some part is shown by the appearance of excess of lactic acid in the urine of Case 1 and Case 2, and by the presence of small quantities of lactic acid in the rectal contents of Case 4. The action does not appear to be great, but is presumably undesirable, and should be minimised. This can be done by washing out the colon regularly and by the

employment of isotonic dextrose (6 per cent.) which is presumably more rapidly absorbed than are stronger solutions, which are diluted in the colon to about 6 per cent. before being absorbed.

We suggest that a useful method of rectal alimentation for an adult would be to give 15 oz. of 6 per cent. dextrose solution in tap-water (10 dr. to 1 pt.) every six hours, which would correspond to 100 grm. dextrose per diem, or 410 Calories. The colon should be washed out daily four hours after the last administration of dextrose with 2 pints of 0.7 per cent. saline solution to which .2 dr. of sodium bicarbonate may be added. If the dextrose solutions are well retained, their concentration may be increased up to 10 per cent. (2 oz. to 1 pt.) making 170 grm. dextrose per diem, or 700 Calories, or 15 oz. of peptonised milk may be substituted for one of the daily doses of dextrose solution.

#### NOTE ON THE PRESENCE OF CREATIN IN THE URINE OF CHILDREN.

Creatin appears to be a normal constituent of the urine of Case 2 in the preceding paper. According to Schwarz, who examined the urine of a normal boy aged 5 under careful conditions as to diet, creatin is absent from the urine of normal children, but present in that of rickety children. The study of the occurrence of creatin in the urine of children in relation to their diet and nutrition appeared, therefore, worthy of pursuit. More recently, however, two papers on the subject have appeared. Rose found creatin to a variable extent in the urine of all children under puberty, but was unable to investigate their diet. Folin and Denis have since confirmed Rose's observations on children whose diets were known, and state that creatin is always present in the urine of children, though in smaller amount when the diet is free from creatin than when it contains meat. Creatin was also present in the urine of children who had never taken meat. We can, therefore, only give our results, and state that we are in entire agreement with these observers.

Case.	Total Creatinin in 100 c.c.	Creatin. Creatinin.	Remarks.
1. Boy aged 1 $\frac{1}{2}$ ...	0.24	0.535	Well nourished. Breast-fed. A little meat
2. Boy aged 2 $\frac{1}{2}$ ...	0.14 0.123	0.48 0.55	Ward diet, "Farinaceous" (no meat)
3. Boy aged 5 ...	0.123	0.079	Well nourished
4. Boy aged 8 ...	0.053	1.15	Well nourished
5. Boy aged 9 ...	0.102	0.072	Moderate nutrition. No meat during 4 preceding days
6. Girl aged 3 ...	0.018	0.82	Well nourished

In conclusion, we wish to thank Dr. Hale White, Mr. Symonds, and Mr. Steward for permission to publish the results of investigations made on patients under their care, and to thank the nursing staffs of Lydia and Addison Wards for their kind co-operation.

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# THE EXTERNAL ABDOMINAL RING, AND ITS IMPORTANCE IN THE RADICAL CURE OF INGUINAL HERNIA.

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By

PHILIP TURNER, M.S.

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SOME months ago I read a paper before the Section of the Royal Society of Medicine for the Study of Disease in Children on the Radical Cure of Inguinal Hernia. The treatment which I advocated was complete removal of the sac through a small incision in the external oblique just above the internal abdominal ring, the external abdominal ring not being divided. At the conclusion of this paper I stated that I had employed a modification of this operation in adults. The object of the present paper is to describe this modification and to give the reasons for its adoption.

In children the essential factor in the causation of the hernia is undoubtedly the presence of the congenital sac which is the result of failure in the obliteration of the processus vaginalis. There is no congenital weakness of the inguinal canal or of the structures which enter into its formation. Before the abdominal muscles have reached their full development, that is, in children in the widest sense of the term, the following principles should be observed in operating for the radical cure of inguinal hernia:

1. The sac, the cause of the trouble, must be completely removed.
2. This must be effected with the least possible damage to normal structures. The abdominal muscles may then be expected to develop normally and a strong inguinal canal to eventually result.

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According to Hamilton Russell the essential cause of a hernia in adults is, as in children, the presence of a congenital sac derived from the processus vaginalis. He denies the existence of a true acquired hernia, or rather of an acquired hernial sac. A hernia which first appears in adult life is, according to this theory, due to the presence of a congenital sac which, though entirely unsuspected, has been present from infancy; the hernia, or "rupture," eventually developing as the result of some gradual or sudden strain which forces some of the abdominal contents into the already present sac. There are certainly many arguments in favour of this view, and, without entering into any controversy, it may be granted that it is true in, at any rate, the great majority of cases.

In adults, however, there is another factor which has to be considered, in addition to the presence of the sac, and that is, that there is very frequently some weakness of the structures which bound the inguinal canal. This is not congenital weakness, but is acquired, and is due to one of the following acquired causes:—

1. It may be due to continued slight pressure of the sac and hernia within the canal interfering with the normal development of the muscles, or later on actually causing them to atrophy. In old-standing large hernias the atrophy may be so excessive that the valvular character of the canal disappears, and examination then shows a gap which may admit several fingers passing directly through the abdominal wall.

2. It may be due to pressure from without by a truss. This is a very important cause of acquired weakness in the muscles which bound the canal, especially in children where a truss may in this way be productive of considerable harm. In patients, both children and adults, who have worn a truss for a long time palpation of the abdomen will often reveal a soft patch in the inguinal region beneath the pad of the truss.

3. Trauma, under which head is included injury to the structures bounding the canal during operation. Now this obviously is a question of the greatest importance as regards a recurrence after operation, and on that account demands careful considera-

tion. Mr. Battle read a paper on this subject before the Medical Society in 1908 (*Lancet*, 1908, vol. ii., page 1601). He found that, as a rule, if the hernia was going to relapse, the return would probably take place within the first twelve months after the operation. He questions, and I think rightly, the usually accepted view that suppuration is the chief cause of recurrence. A hernia may certainly relapse though the wound has run a perfectly aseptic course, and conversely in many cases which become infected there is no recurrence. Recurrent hernias fall into two groups. In the first the sac reappears following the course of the spermatic cord and projecting from the external ring or even extending down into the scrotum, much as it did before the operation. These are probably, as Mr. Battle points out, due to imperfect removal of the sac at the original operation. The sac was not ligatured sufficiently high up, or possibly the ligature slipped or became displaced as the result of some strain after the operation such as severe post-anæsthetic vomiting. In the second and more frequent group we find a splitting up of the aponeurosis of the external oblique from the external ring in an upward and outward direction, *i.e.*, in the course of the inguinal canal, and a general bulging forwards of the scar. These can scarcely be regarded as true recurrences of the original hernia, but are rather analogous to the ventral hernias occasionally seen after appendix operations where drainage has been necessary, and are due to the same cause, *viz.*, injury to the abdominal wall in the region of the incision leading to weakness and subsequent yielding of the scar. To avoid recurrences of this nature it is of the greatest importance to operate in such a way as to cause the least possible injury. A hernia after an appendicectomy between attacks is now a rare occurrence. This is owing to the fact that the muscles of the abdominal wall are divided in the direction of their fibres, or the peritoneal cavity is opened by a valvular incision through the rectus sheath. The splitting up of the aponeurosis mentioned above starts at the upper boundary of the external ring which thus becomes of very great size and assumes

a triangular shape owing to separation of the pillars. This structure, and especially its upper margin where the splitting starts, would thus appear to be of the greatest importance in the prevention of this variety of the recurrence of the hernia.

It is now necessary to consider briefly the anatomy of the external abdominal ring. It is usually described as a cleft in the aponeurosis of the external oblique, triangular in shape, the base being formed by the crest of the pubis, the outer and lower boundary by Poupart's ligament (the outer pillar), the inner and upper boundary by that part of the aponeurosis attached to the crest and symphysis of the pubis (the inner pillar), and the apex by the meeting of the two pillars above. Above the ring the two pillars are connected by a number of transverse fibres which are continued down to form a thin tubular sheath enclosing the spermatic cord, and known as the intercolumnar fascia. This account is correct, except that it does not give a sufficiently good account of the upper limit of the ring which we have seen is of such importance in recurrence of the hernia after operation. It is easy to satisfy one's self that the upper limit of the ring is not an angle formed by the meeting of the two pillars. All that is necessary is to examine a patient with a hernia, or, indeed, a normal individual, lying down on a couch, by invaginating the scrotum and sweeping the finger round the margin of the ring. Such an examination will show that the ring is bounded above by a definite rounded margin, and if the examination be repeated with the patient in the erect position it will be found that this margin is subject to very considerable tension. Examination of a number of external abdominal rings at hernia operations shows that though the arrangement varies there is always a more or less complex arrangement of fibres to strengthen this important spot. Figs. I. and II., drawn diagrammatically from actual rings, show a typical arrangement. It will be seen that in addition to a strong band of intercolumnar fibres there are also strong decussating bands passing from the inner pillar to the lower part of Poupart's ligament and from the upper part of Poupart's ligament to the lower part

of the internal pillar. In most cases these are of much greater importance in securing the strength of this part of the ring

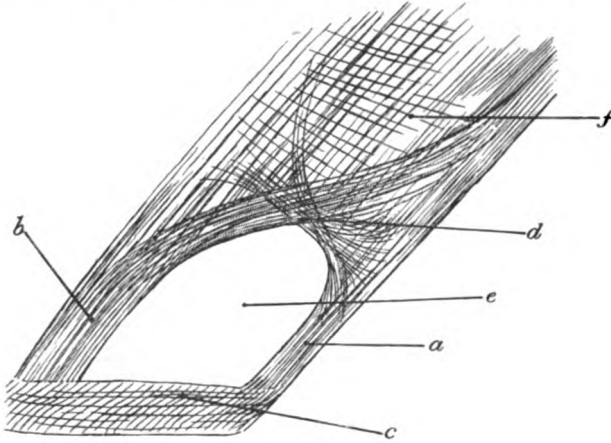


FIG. 1.

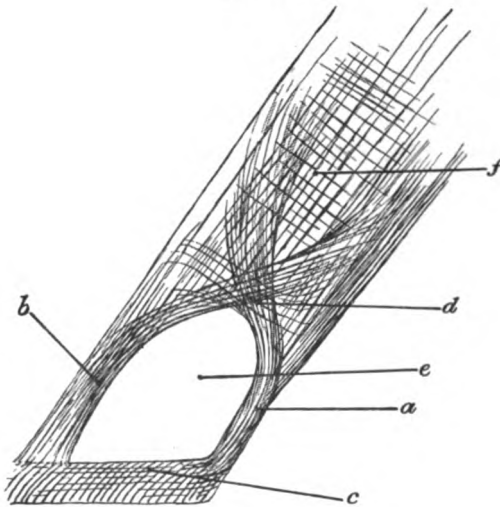


FIG. 2.

*a.* Poupart's ligament (external pillar). *b.* Internal pillar. *c.* Crest of the pubis. *d.* Intercolumnar ligament. *e.* External ring. *f.* Intercolumnar fibres.

than the intercolumnar fibres, and they also are responsible for the rounded character of the upper margin. Of such importance



is this upper boundary of the ring that it seems to be worthy of a definite name. I would suggest "intercolumnar ligament" or "intercolumnar band," and shall thus refer to it in the succeeding remarks.

The structure of this ligament varies somewhat in different cases. Thus, in some rings there will be a well-marked band of fibres passing from the inner pillar to the outer, while in others the reverse is the case. Occasionally the transverse fibres will be best marked, and then these will often be seen to end by turning upwards or downwards to become continuous with the longitudinal fibres of the two pillars. Above the intercolumnar ligament the fibres of the aponeurosis of the external oblique will be seen to have much the same direction as the axis of the inguinal canal, though a few feebly-marked transverse fibres may be traced as high as the level of the anterior superior spine of the ilium. This complex arrangement of the fibres which go to form the intercolumnar ligament is a proof of the tension to which this part of the ring is subjected and which can be easily verified, as mentioned above, by a digital examination of the ring in the living subject. The disadvantage of dividing muscles or aponeuroses, except in the direction of their fibres is well known. If divided transversely the subsequent tension is likely to cause weakness or yielding of the scar, while if split in the direction of their fibres and afterwards carefully sutured such a result is very unlikely. The same is true of the intercolumnar ligament. If once divided this structure cannot, even by the most careful suturing be reconstructed so as to be as strong as it originally was. The normal tension in this situation is then apt, when active life is resumed after the operation, to make the upper boundary of the ring give way and to start some splitting or bulging of the anterior wall of the canal. If, on the other hand, the intercolumnar ligament has been preserved, this splitting and bulging will not occur, for the undamaged ligament is quite able to withstand the tension. It will thus be seen that in an operation for the radical cure of inguinal hernia the preservation of the external abdominal ring is a matter of great importance.

It will now be necessary to consider the nature of the acquired weakness of the canal. This may affect both the anterior and the posterior walls. The posterior wall is formed partly by the conjoined tendon, but in the outer half of its extent this structure is absent, so that we have here only the transversalis fascia, the extra-peritoneal tissue, and the peritoneum. As the result of the presence of the hernia the conjoined tendon may have imperfectly developed, or may have become thinned out so that the extent of the weak portion of the posterior wall formed by the transversalis fascia is much increased. The anterior wall is formed throughout its extent by the aponeurosis of the external oblique, and in its outer third also by the internal oblique. The acquired weakness of the anterior wall is chiefly seen in the increased size of the external abdominal ring, but also to a slight extent in the thinned-out condition of the lower border of the internal oblique which no longer, as it normally should, closely encircles the spermatic cord as it emerges from the internal ring. In advanced cases the valvular arrangement of the canal disappears, so that the large external ring is opposite the increased weak portion of the posterior wall. There will then be a direct gap in the muscular structures of the abdominal wall through which several fingers can be introduced. It is thus seen that in cases of hernia in adults where secondary weakness has appeared, removal of the sac alone is unlikely to be sufficient to cure, or rather to prevent recurrence of, the hernia. In these cases it will be desirable to make some attempt to strengthen the weakened structures.

It is always desirable to estimate the amount of acquired weakness in any case in which a radical cure is advised. This may be done by a digital examination of the external ring and of the inguinal canal. The lower part of the scrotum is invaginated by the forefinger which is passed upwards until the crest of the pubis is felt. The external ring will be found immediately above this. The size of the ring is then estimated by sweeping the finger round its margin, when the rounded upper border formed by the intercolumnar ligament is also noticed. The nor-

mal ring should just admit the tip of the forefinger. If the tip of the finger can be made to traverse it and enter the canal, the ring is certainly enlarged. If the tip of the finger be pressed backwards through the ring the conjoined tendon will be felt. If the ring is slightly enlarged the thin sharp edge of this structure will be felt, while if the canal is seriously weakened the finger can be made readily to impinge upon that part of the posterior wall formed by the transversalis fascia. By this examination it is possible in most cases to decide whether it will be necessary to strengthen the canal or whether simple removal of the sac will suffice.

The operation is now carried out on the following lines. An incision is made, commencing just external to the spine of the pubis and extending upwards and outwards just above and parallel to Poupart's ligament. The aponeurosis of the external oblique is exposed throughout this incision. The external ring, which is just at the lower limit of the skin wound, is not necessarily exposed though the intercolumnar ligament is identified. The aponeurosis is divided for a distance of an inch in the direction of its fibres, commencing immediately above and to the outer side of this structure. The internal oblique is thus exposed and probably, in the lower part of the wound, a small area of the posterior wall formed by the transversalis fascia. The interior of the canal can now be displayed by introducing a small hook-shaped retractor through the wound in the external oblique and drawing the lower margin of the internal oblique upwards and outwards. The spermatic cord, covered by the cremaster, is now seen. The cremaster and the infundibuliform fascia are now torn through with the help of two pairs of dissecting forceps, and the spermatic cord and sac drawn forwards through the opening in the aponeurosis and retained in this position by a blunt dissector or forceps passed beneath them. The structures of the cord are now spread out as much as possible over the dissector or the finger when the edge of the sac readily comes into view. It is secured by a pair of Spencer Wells forceps which just grip the edge. Still keeping the cord spread

out, the sac is completely separated in a transverse direction from the vas, veins, and other structures, by means of a blunt dissector. It is now possible to introduce the finger between the sac and cord at the site of this separation. The sac is now firmly grasped, and with a few touches from a blunt dissector, aided by wiping movements with a swab or piece of gauze, one completes the separation in an upward direction as far as the internal ring. When this is done the lower end of the sac is strongly drawn upon, and by the same means the lower part of the sac is separated from this portion of the cord. The most difficult part of the separation is usually the lower end of the sac which, by strong traction on the isolated portion, is drawn up from below the external ring. In an old-standing hernia where the sac is adherent and extends well down into the scrotum very strong traction may be necessary to bring the lower end of the sac into view. It is interesting however, to note that, owing to the strength of the intercolumnar ligament, the external ring is never split open by these manipulations. The arrangement of the fibres of the intercolumnar ligament prevents this from happening. Some splitting may take place in an upward direction, but, as this will be simply separation of the fibres of the aponeurosis, weakening of this structure will not result if the rent is subsequently carefully sutured. Separation of the sac, even when it extends down to the tunica vaginalis, may be effected in this way without division of the intercolumnar ligament, for traction on the upper freed portion of the sac will draw the testicle up so that it will present at the external ring.

When the whole sac is completely separated it is strongly pulled upon by an assistant so as to draw peritoneum down from the abdomen and to expose the extra-peritoneal tissue above the internal ring. At this point it is transfixed, ligatured, and the stump then fixed above the level of the internal ring by threading the two ends of the transfixing ligature and passing them through the internal oblique muscle.

The posterior wall of the canal is now examined. If the internal oblique is well-developed and the conjoined tendon forms a good extent of the posterior wall of the canal, as is frequently the case even in old-standing hernias, it is best to leave well alone. There is every prospect that complete removal of the sac will be successful in producing a radical cure. Under these circumstances any attempt to suture the conjoined tendon to Poupart's ligament will not be productive of good, and may, indeed, by fraying out or strangulating portions of these structures actually cause harm. In those cases, however, where the internal oblique and conjoined tendon are badly developed or are lax and thinned out, such sutures will probably strengthen the posterior wall. They may be applied as follows without dividing the intercolumnar ligament :—

Two pairs of tissue forceps are made to encircle the whole of the structures of the cord. These retract and raise the cord at the upper and lower ends of the wound respectively, and thus afford a good view of the posterior wall of the canal. It is now possible to see the conjoined tendon distinctly, and two or more mattress sutures are inserted between it and Poupart's ligament, as in Bassini's operation. When this is accomplished the cord is replaced in the canal. If so much of the cord has been drawn out in the course of these manipulations that its replacement is difficult, this may be effected by an unsterilised assistant putting his hands beneath the towels, grasping the testicle and drawing this well down into the scrotum, thus also drawing the spermatic cord into position. When this is done the internal oblique falls back into its place, and, as a rule, this muscle will extend throughout the whole of the incision through the external oblique, so that the operation will have been effected through an incision which scarcely opens the anterior wall of the canal. The small incision in the aponeurosis of the external oblique is now carefully and accurately sutured with fine catgut.

The question now arises, should anything be done to an unduly large external ring? I think that even a very large external ring is best kept intact so as to preserve the intercolumnar liga-

ment, but in these cases there can be no harm in putting a few sutures to draw the pillars of the ring together. Indeed, it is probable that such sutures will do good.

The operation can certainly be carried out in this way in most young adults with the greatest ease, but it may be asked, is it always possible to preserve the ring intact? I think it must be sacrificed in the following cases:—1. Strangulated hernias. 2. Irreducible hernias. 3. Very large old-standing hernias or hernias with very thickened and adherent sacs. Of course, in any case if the circumstances seem to demand it, the intercolumnar ligament can be divided and the ring be opened up at any time during the course of the operation.

A radical cure of inguinal hernia thus carried out ensures complete removal of the sac with the least possible amount of damage to, and weakening of, normal structures, and at the same time allows of the application of sutures to strengthen both the anterior and posterior walls of the canal should these be considered necessary. Additional advantages are that the separation of the sac is commenced at the point where separation is easiest, viz., just below the internal ring, and that the inner limit of the skin incision is about an inch further from the groin than in the ordinary Bassini's operation.

No truss is worn by the patient after the operation. It is interesting to examine patients a few weeks after the operation when carried out in this manner. On invaginating the scrotum as described above, the external ring feels usually quite normal, and it is possible to introduce one's finger and explore the canal, which usually shows but little thickening; it is also unusual to find any alteration in the spermatic cord. I have systematically employed this modification for about the last year—too short a time to investigate the occurrence of frequency or recurrences, but hope to do this on some future occasion.



# TWO EXAMPLES OF OPALESCENT SEROUS FLUIDS.

By

J. H. RYFFEL AND E. M. MAHON.

OPALESCEMENT or milky ascitic fluids have recently been studied by Mackenzie Wallis and Schölberg.\* They divided these fluids into two groups: chylous, in which the opalescence was due to the presence of chyle from the lymphatic system, and pseudo-chylous, in which chyle was not present; thus abandoning the previous classification of chylous, chyloform, and milky, non-fatty effusions, because the opalescence of the chyloform group is mainly due to the same cause as that of the milky, non-fatty group.

The principal characteristic of the pseudo-chylous effusions is that, besides a variable amount of fat globules, they contain minute particles which show Brownian movement, and to which the opalescence is really due. These particles are not removed by ordinary filtration, nor do they form a creamy layer on standing or centrifugalising. Such a creamy layer often forms in these fluids, but it is due to fat globules, which are not the cause of the opalescence, as this persists after they have separated. The minute particles can be removed by filtration through a Pasteur candle, so that the filtrate becomes perfectly clear. They are also largely removed by dialysis followed by filtration and by half saturation with ammonium sulphate followed by filtration. In the latter case the precipitate forms an opalescent

\* Mackenzie Wallis and Schölberg. *Quarterly Journal of Medicine*, III., 1910, 301, and IV., 1910, 151.



liquid with water similar to the original fluid, and yields lecithin on extraction with hot alcohol. It can also be shown to contain globulin. The authors conclude that these minute particles, to which the opalescence of the fluids is due, consist of a complex of lecithin and globulin, which is precipitated by the same reagents as is globulin. Lecithin alone does not produce a similar opalescence.

The cause of the presence of these particles is unknown, but they are of interest in the indication they give of the manner in which fatty bodies are held in solution in the fluids of the body. The analyses of Abderbalden have shown that there are in blood serum considerable quantities of lecithin, cholesterol, and fatty acids. Part of the fatty acid is present as fat, but by no means all. Hardy found esters of cholesterol and fatty acids associated with all the different fractions of protein obtained from blood serum. When either lecithin or cholesterol ester was added to a globulin solution it dissolved at first to a clear solution, but with more of the fatty body the solution became opalescent. The opalescence of the blood in diabetic lipæmia appears to be largely due to a similar cause, as Wilson and Williams\* and Roaf and B. Fischer found a cholesterol ester present in the blood in these cases. In normal blood and tissue fluids, therefore, the lipoids present form in association with the proteins a colloid solution, the particles of which are so small that they pass through a Pasteur candle and do not cause the solution to be opalescent. When, however, the lipoids are in excess in comparison with the proteins, the particles are larger, so that they cause opalescence, are visible under the microscope, and do not pass through the pores of a Pasteur candle. In the liquid of pseudo-chylous ascites the lipoids are frequently not present in greater quantity than in serum, but the protein is very much less (usually not more than two per cent., and even as little as 0.15 per cent. is recorded), so that the cause of the opalescence is the excess of the lipoids in comparison with the protein.

\* Wilson and Williams. *Biochemical Journal* II., 1907, 20.

According to Wallis and Schölberg the liquid of pseudo-chylous ascites is characterised by (i.) the presence of these particles consisting of lecithin and globulin, (ii.) the relative absence of cholesterol, (iii.) the relative absence of fat globules, (iv.) low specific gravity, (v.) low solid content, (vi.) low protein content, (vii.) low actual fat content, and (viii.) lack of odour.

On the other hand, the liquid of chylous ascites is characterised by (i.) the relative absence of lecithin and globulin, (ii.) the presence of cholesterol, (iii.) the presence of fat globules, (iv.) higher specific gravity, (v.) higher solid content, (vi.) higher protein content, (vii.) higher fat content, and (viii.) characteristic odour.

They collected 173 cases during the last fifty years, of which 71 were probably pseudo-chylous. The diseases in which milky fluids of either type most commonly occurred were tumours of the abdominal organs, tuberculosis, cirrhosis, and nephritis. Those associated with nephritis were practically all pseudo-chylous, while those associated with obstruction of the thoracic duct or distension of the chyle vessels were mostly chylous.

Case 1.—A well-developed woman, 26 years of age, died in a uræmic condition with œdema and ascites as the result of an attack of acute on chronic nephritis. Thirteen days before death about eight litres of opalescent ascitic fluid were withdrawn. Post-mortem, 800 c.c. of opalescent ascitic fluid were obtained. The pleural cavity contained about 500 c.c. of fluid, which was not opalescent. The kidneys were large (556 grm.) and contained much blood. The cortex was well differentiated and bore the normal proportion to the medulla. Microscopically the tubules were very markedly dilated and their epithelium was markedly flattened. The intertubular connective tissue was considerably increased.

The first specimen of ascitic fluid had a specific gravity of 1007. It was alkaline, reduced Fehling's solution, and contained a few leucocytes and two parts per thousand of albumen. The opalescence was due to minute particles, which did not separate on centrifugalising.

The post-mortem specimen was rather more opalescent. It did not become clearer on filtering. It was alkaline, specific gravity 1007, contained three parts per thousand of albumen. No coagulum was obtained on heating until acetic acid was added. Sugar was present. Lymphocytes, but no fat globules, were present.

With a one-sixth objective minute granules showing Brownian movement were seen. No creamy layer formed on standing, but continued shaking with ether caused the liquid to become somewhat less opalescent. The addition of caustic soda rendered the extraction with ether more complete, but still the liquid was not completely cleared. On dialysis and on half saturation with ammonium sulphate a precipitate formed. The filtrate from the latter was practically clear, while the precipitate formed with water a white opalescent fluid which passed through the filter.

The total solids amounted to 2.05 per cent., and the ether extract of this to 0.116 per cent. of the original liquid. The ether extract of the alcoholic extract of the liquid gave a considerable precipitate on the addition of acetone after concentrating the extract, which contained phosphoric acid, and was, therefore, some form of lecithin. Cholesterol was not present.

These reactions are all typical, so that the case was clearly one of pseudo-chylous ascites associated with nephritis.

Case 2 was a man 59 years of age, who had for a year previously suffered from enlarged glands in the neck, axillæ, and groin, with œdema of the right limb. A month before death a milky fluid was withdrawn from the *pleural* cavity. Post-mortem, this fluid was found in both pleural cavities amounting in all to 1600 c.c. There were sarcomatous deposits in both lungs, in the posterior mediastinum, about the abdominal aorta, and in one kidney.

The first specimen of pleural fluid had a specific gravity of 1017, and was alkaline. Filtration scarcely reduced the opalescence. The total solids amounted to 7.52 per cent., and the ether extract of this, which contained a trace of lecithin, to 0.16

per cent. of the original liquid. Some yellow fat globules were present, together with a large number of minute particles showing slight Brownian movement.

The post-mortem specimen had the same general characters as the earlier specimen. It contained 2.8 per cent. of protein, and coagulated readily on heating. The total solids amounted to 7.33 per cent., and the ether extract of this to 0.14 per cent. of the original liquid. It formed a creamy layer on standing, but the lower part of the liquid remained opalescent. On shaking with a large excess of ether the liquid became much clearer. It filtered perfectly clear through a Pasteur candle.

On filtering off the precipitate obtained on half saturating with ammonium sulphate, a perfectly clear filtrate was obtained, whilst the precipitate formed with water an opalescent liquid, which was unaltered by filtration. The ether extract of the alcoholic extract of the liquid contained only a trace of lecithin.

Finally, 300 c.c. of the liquid were repeatedly shaken with large volumes of ether. The ether was evaporated to small bulk and acetone added. A precipitate resulted which amounted to 0.08 per cent. of the original liquid. On evaporation of the acetone solution, liquid fatty material amounting to 1 per cent. of the original liquid was obtained, so that this method of extraction was much more complete than that used above of extracting the dried solid with ether in a Soxhlet apparatus, showing that the principal lipoid present was probably not fat as such.

The precipitate obtained above by the addition of acetone contained only a trace of phosphoric acid, gave a good Liebermann and Burchard test for cholesterol, and was slightly soluble in cold acetone, so that it was evidently mainly not lecithin. It was soluble in hot alcohol, and crystallised on cooling in minute hairs radiating from a centre. On hydrolysing with alcoholic potash the solubility of the material was much increased, and typical crystals of cholesterol were obtained which gave characteristic tests. The material, therefore, consisted mainly of a cholesterol

ester. This cholesterol ester was also present in the original acetone soluble part, as this also gave the cholesterol test and yielded crystals of cholesterol on hydrolysis.

The minute particles which were the principal cause of the milkiness of this liquid, and which showed similar properties to those of the particles that occur in pseudo-chylous ascites, consisted, therefore, of a complex of a cholesterol ester, probably with globulin.

The amount of protein present, the presence of cholesterol, and the practical absence of lecithin would point to a chylous origin for the fluid, but these particles are not such as are described in chylous ascites.

Our thanks are due to Dr. Hale White and to Dr. Hertz, and also to the Demonstrators of Pathology of Guy's Hospital for permission to publish these cases.

# THE RESULTS OF OPERATION FOR GLAUCOMA.

AN ACCOUNT BASED UPON THE EXAMINATION OF  
79 CASES.

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COMPILED BY

ALAN H. TODD, M.B., B.S., B.Sc.

(for the Charles Oldham Prize at Guy's Hospital).

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THE object of this paper is to find out what measure of success attends the operative treatment of glaucoma, and the factors which affect it. For this purpose it is necessary to have an accurate record of the condition before treatment, so only those cases have been studied in which the needful data have been preserved in the reports. A circular-letter was sent to all these patients, requesting their attendance at the hospital for examination. Rather less than 30 per cent. of replies were obtained, but in a few instances the details of the post-operative course of the case have been entirely derived from the out-patient letters. The statistics have, therefore, the value of a consecutive rather than of a selected series, the elimination of certain cases being the result of accident, and not of design.

The cases fall easily into three classes, namely, Primary Acute, Secondary, and Chronic. It will be seen that they are very different, not only in their causation, but also in their subsequent history.

## I.—ACUTE.

Of 15 cases, 3 occurred in men, and the remainder in women. The average age of the men was 55.5 years, and of the women, 60 years. The youngest patient was 49 years old, and the oldest was 74. All underwent iridectomy. In 6 cases the result was decidedly good, in 5 it was bad, and in the rest it was satisfactory. The details are given in the case-reports in the appendix, but their important features may be cited here:—

*Good Results—*

1. On admission, T+, optic disc cupped, no P.L. After iridectomy, field became normal, vision was 6/6 and J<sub>1</sub>. Three years later, vision was 6/9 and J<sub>1</sub> with glasses.

2. Seen six days after onset, T-3, both fields full, V=H.M. Vision=6/18 and J<sub>4</sub> after operation, and 6/6 and J<sub>1</sub> 2½ years later. Both fields were full; *vide* perimeter charts.

3. Came on after influenza three weeks previously; atropine was put in. On admission, T+2, field much contracted, V=H.M. at 1 foot. After operation, V=6/36 without glasses, and patient is able (3 years later) to read, sew, or knit without any difficulty or pain. The result would probably be still better, as to vision, if there were not some associated opacity of the lenses.

4. Three weeks' history. T-2, some nasal contraction of field. After iridectomy, V=3/60, O.D. not cupped, Tn. Two and a half years later, V=6/9 and J<sub>1</sub> with glasses, and the fields only slightly contracted on the nasal side. There were no signs or symptoms of increased intra-ocular pressure in the eye operated on.

5. Ten weeks' history, and atropine instilled. On admission, T+. After iridectomy, Tn, field much contracted, V=4/60. Seventeen months later, V=6/60 without glasses, and 6/36 with them.

6. Fourteen days' history. On admission, T+3. Three days later it was noted that the iridectomy wound was leaking, and there was much lens-opacity. Now (2¼ years later) patient

writes that his distance and near vision are good in each eye, and that he has no pain, coloured rings, headache, or blurring of vision at any time.

It will be noticed that in the first three of these cases very good results were obtained, although the vision was only H.M. on admission (Case 3), or even nil (Case 1). There is nothing common to them all whereby one may explain the marked success that attended treatment in their case. The patients were no younger than the rest, nor was the intra-ocular tension by any means low. They were seen, however, fairly early, and there is in each report one feature at least that points to a good prognosis. Thus, in Case 1, vision returned very rapidly after the operation, and the field showed no impairment. It seems reasonable to assume that the tension was successfully lowered before any destructive change had taken place within the eye. In Case 2, though the tension was high and the vision reduced to a minimum, the fields were full before operation. It must be remembered in this connection that the field of vision is of far greater prognostic importance in glaucoma than the acuity. Cases 3 and 5 are examples of the misuse of atropine. The striking feature about them is that vision was ultimately restored to a large extent, although only after a considerable lapse of time. In the latter instance operation on the painful eye was followed in eight days' time by acute glaucoma in the other eye. Vision was reduced to 6/60, but the field, after iridectomy, remained almost normal. Seventeen months later, V=6/18 with glasses. Case 4 was instructive in several ways. When the patient left the hospital her vision was only 3/60, but her field was not much diminished, and her tension was normal. With the lapse of time, the vision returned practically to normal. On examination it was discovered, however, that there was some early nasal contraction of the opposite field, which had formerly been full, as well as some pain in the eye. Vision was still perfect, and the tension was not raised on the day when the patient was seen. Nevertheless, there was definite evidence of early glaucoma in the eye, and the patient was advised to



undergo iridectomy, experience having shown that in chronic cases the results are only really satisfactory when they are treated early. She refused to entertain the suggestion, however, preferring to "wait until it gets bad, because it was so easily done before." Apart from difficulty of diagnosis, this reluctance of patients to submit to an operation which seems to them, even after explanation, to be less a matter of necessity than of possible expediency, is one of the chief reasons why the prognosis in chronic disease is so poor. Many examples of this will appear later on in this paper. Case 6 has a topical interest of its own in that a leaking wound resulted from the incision for iridectomy, with excellent result. In spite of some lens opacity the patient can see well, and has no further symptoms of increased tension.

#### *Bad Results—*

11. Five weeks' history. After iridectomy the patient could only see some figures on a watch. Now the pupil is non-reacting, there is lymph on the back of the cornea as well as deep in the anterior chamber, and there is a mature amber-coloured cataract. V=fingers at 1 foot. There is early glaucoma in the opposite eye.

12. This may be really a case of chronic disease, for the history is that 18 months ago the sight was "weak." Then came an attack of influenza, after which the eyes became suddenly bad; then the sight began to decline rapidly. Whether the early "weakness" was due to presbyopia or to glaucoma cannot now be determined, but the case was classified at the time as one of subacute disease. On admission, the iris was atrophic, the tension  $+3$ , and the field much contracted. V=6/18. After operation, when the lens-capsule was wounded, the pain persisted, T $+2$ , and V=P.L. Iridectomy was repeated, with sclerotomy, and later on cyclodialysis was performed. At present, V=P.L. The other eye was also glaucomatous on admission, with P.L. only: sight was not improved by iridectomy.

13. Two months' history. T+3; disc cupped and atrophic; no P.L. after operation. In the other eye, after iridectomy, V=3/60.

14. Patient with hæmorrhagic retinitis; acute glaucoma after some illness. On admission, no P.L. and T+2. Pain persisted in spite of operation, and excision was performed.

15. One month's history. Lenses sclerosed. T —. No P.L. After iridectomy pain continued, vitreous was extruded, T+2, and no P.L.; excision.

Of these five cases, No. 12 can probably be excluded from the present list, as being really chronic. The same may apply to No. 13, for one can hardly imagine that marked cupping and atrophy of the disc could occur in a genuinely acute case. Besides this, there was definite glaucoma, simultaneously, in the other eye, a most unusual event in the acute class, though the appearance of the disease first in one eye and then in the other, in rapid succession, is not uncommon. In No. 11 the present deficiency of vision is sufficiently explained by the cataract, but the bad result following the operation has still to be accounted for. From the appearance of the eye now it would seem that some infection must have occurred at the time, and this in itself would be a sufficient explanation of the disappointing result. Case 14 was apparently due to a primary hæmorrhage, and did badly, as such cases generally do. Presumably it is very difficult for the highly albuminous fluid to filter away, so that the tension remains high for some time; in addition, its opacity prevents the transmission of light, so that perception of light is slow in returning, if, indeed, it returns at all.

Given, then, a case of glaucoma within a month of its onset, there seems to be no reason to anticipate other than a good result from iridectomy; for, of the cases that did badly, one was probably due to infection, which is a preventable evil, and the others to hæmorrhage and to extrusion of the vitreous, which are unfortunate accidents, and not a part of the glaucomatous process. There is a great difference, however, between hæmorrhage taking place before operation, and causing glaucoma, and that which sometimes follows operation, and is caused by it. The

latter is a very serious complication, for it considerably reduces the amount of vision ultimately obtained, and not infrequently necessitates the removal of the eye altogether on account of pain and blindness. The danger is a very real one, ever present in cases of high tension (+3), and especially in persons afflicted with arterio-sclerosis. Any means, therefore, of avoiding such a catastrophe would be of the greatest value. What is needed is a method of reducing the tension gradually so as to avoid a sudden strain on the walls of the blood vessels. It is possible that this might be found in preliminary paracentesis, followed by iridectomy or sclerectomy, a measure that is not new, but does not appear to have been extensively adopted at Guy's.

## II.—SECONDARY.

Of 28 cases, 13 occurred in women and 15 in men. The average age of the men was 51·2 years, and of the women was 48 years. The youngest patient was 19 and the oldest was 76. The various causes at work were:—

Foreign body, injury, or hypopyon—

Cases 16 to 19	...	...	...	...	...	4
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Intra-ocular hæmorrhage—

Case 20	...	...	...	...	...	1
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Dislocation of lens—

Cases 21 to 24	...	...	...	...	...	4
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Iritis—

"Rheumatic," Cases 25 to 28	...	...	...	...	4
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Syphilitic, acquired, Case 29	...	...	...	...	1
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Syphilitic, congenital (?), Case 30	...	...	...	...	1
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Old and recurrent, Cases 31 to 43	...	...	...	...	13
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Total	...	...	...	...	...	28
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It is necessary to consider each of these four groups separately. The four cases due to *foreign body, injury, or hypopyon* all occurred in men, three of whom were elderly. In the first, a corneal ulcer perforated, a staphyloma formed, and the anterior

chamber was obliterated. The tension rose to  $+2$  or  $3$ ; iridectomy was performed, and the tension fell to  $+1$ , but the lens became opaque (possibly from a prick of its capsule during the operation), and the eye is now blind, though painless. In the second there was an old history of raised tension in each eye, and on admission the left lens was found to be dislocated, the anterior chamber was infected, and there was no P.L.;  $T = +2$ . Iridectomy failed to cause improvement, and the eye was excised. The third was very similar; the eye was blind and was removed. In the fourth case  $V = P.L.$  and  $T = +1$ , and continued raised after sclerotomy; excision was performed.

From these four cases it appears that the prognosis in glaucoma due to a foreign body or its effects is very bad; not only is sight completely destroyed, in many instances, but the eye has often to be removed on account of persistent pain.

The *intra-ocular hæmorrhage* occurred in a woman who had a cataract removed from the same eye a year previously. Tension was  $+2$ , and was not relieved by paracentesis; iridectomy was successful in reducing it to normal, and the pain disappeared. There is no note as to the vision.

*Dislocation of the lens* is a disastrous condition when associated with glaucoma. Of four patients, two had to submit to enucleation, one lost all perception of light (this resulted from panophthalmitis), and the other retained only P.L. The sight of the opposite eye was unimpaired in three out of the four cases; the last was an instance of double dislocation.

Four out of nineteen cases due to *iritis* gave a history of rheumatic fever. Whether this is an example of coincidence or of cause and effect cannot now be discussed, though it seems unlikely that 5 per cent. of patients with acute rheumatism would have suffered from iritis unless there were some causal relationship between the two events. Two patients lost P.L., but their eyes remained painless. In one instance (Case 25) there was calcification of the lens-capsule, and in the other (Case 28), not only was the lens somewhat opaque, but later on the pupil was completely covered by the iris, which was drawn

up and incarcerated in the scar. Case 26 is that of a man who had had recurrent attacks of iritis for many years. On admission the left eye had been blind for four years,  $T = +2$ , and  $V = P.L.$  In the right eye tension was only  $?+$ , and  $V = 6/36$ . Iridectomy was performed on each eye, with this result: right eye, tension normal, field almost full, but vision fallen to  $6/60$ ; left eye, tension normal,  $V = H.M.$  The result is more satisfactory when the symptoms are of more recent onset. In Case 27 there had been sharp pain for a fortnight when the patient came under treatment, and vision was quite gone. The iris was bombé.  $T = N$ . After iridectomy vision was  $4/60$ , and two years later the tension was normal and  $V = 6/24$  and  $J_{16}$  with glasses, though the field showed some general contraction.

Early operation also produced a good result in the one case of acquired *syphilis*, i.e., No. 29. The patient had had three previous attacks of iritis following primary syphilis 16 years before, which had only been treated for five months. The glaucomatous symptoms had been present for fifteen days when he was admitted, with  $T+$ ,  $V = H.M.$ , posterior synechiæ, and old lymph in the pupil. When seen, three years after operation, the fields were full, there were no symptoms of increased tension, the discs were normal, and vision was  $6/9$  and  $J_2$  with glasses. There were no indications of glaucoma in the other eye.

(With regard to this case, it may be of interest to note that examination of the eyes and a change of glasses were followed by another slight attack of iritis, though it was not accompanied by increase of tension.)

The last thirteen of the secondary cases may be considered together. They are all due to *old and recurrent iritis*, the cause of which is not specifically mentioned in the reports. Operation was performed in every case but one, but the results were disappointing. Three only showed any improvement of vision, and this was of so slight a degree as to be of little practical use; one (No. 43) became actually worse, her sight decreasing from  $6/9$  with glasses to a condition in which she "cannot see across the street." The patient says that her vision failed rapidly after

operation, but it is only fair to mention that she had small nebulæ on admission, and that these were found on re-examination to have increased considerably in density. However, the fact remains, whatever the explanation may be, that iridectomy was followed by a diminution of vision. The important features of these thirteen cases are given below in tabular form:—

Case.	Sex.	Age.	Duration.	Right Eye.		Left Eye.	
				Before H.M.	After. 6/60	Before.	After.
31	F	32	4 months			6/5	
32	F	26	4 years	N.		*	
33	M	36	5 weeks	6/9		0	* Excised
34	F	50	1 year	6/36	* 6/12; J <sub>4</sub>	6/60	* H.M.
35	M	46	9 months	6/9		P.L.	*
36	F	72	1 year	*	6/60	*	
37	F	61	—	6/24		F. at 6ft	
38	F	67	<1 year	6/18	*	6/18; J <sub>12</sub>	
39	M	50	3 years	No P.L.	* Excised	6/12	
40	M	76	—	No P.L.	* 6.60	Fair	* 6/36; J <sub>12</sub>
41	M	46	(?) years	No P.L.	*	Good P.L.	
42	F	31	15 years	No P.L.	*	H.M.	*
43	F	51	30 years	6/9	* 6.60 (?)	6/36	*

\* indicates operation on this eye.

*Notes.*—Case 33 may, perhaps, be of much greater duration than at first appears, for his history is that his left eye was blinded by an injury 21 years before, but only became painful five weeks before admission. In No. 38 iridocyclitis and glaucoma came on three years after cataract-extraction. Paracentesis did nothing towards relieving the tension, muddy iris, etc. No. 40 would probably have given a better result than appears above had he not developed cataract, for it is noted that, on discharge, there was no cupping of either disc, and the vision was slowly improving.

Few notes are given in the reports of the effect of iridectomy on the other symptoms, but three patients (34, 39, and 43) complained of persistence of pain; only in one was it severe (39), and in this instance the eye was excised. One patient said that though her eye was painless she invariably saw coloured rings round lights after dark, and another had pain in the non-operated eye due to chronic glaucoma.

It is a striking feature of this collection of cases, due to iritis, that vision was generally considerably impaired when the patients first sought advice. Frequently the disease is of several years' standing, and secondary changes unite with the glaucoma in rendering the prognosis as to vision a poor one. Opacity of the cornea, lens, or lens-capsule is a frequent concomitant. The age of the patient, the duration of the attack, the amount of increase of the intra-ocular tension, and the shape and mobility of the iris—all these vary considerably in the cases quoted above, but it is impossible to show a fixed relationship between these factors and the ultimate prognosis as to vision. Rather would it seem that this depends on some personal peculiarity, some special vulnerability of the tissues by glaucoma, for as much harm is done in some cases in four weeks as would result in another person, apparently identical, in four years. From the patient's point of view, however, the exact diagnosis of the cause of his loss of sight is of little moment; he does not care whether it is due to iritis, to glaucoma, or to a nebula. What he wants to know is, how much vision he may reasonably hope to recover, and, taking the above table of cases, which shows the results of the complications as well as of the glaucoma itself, it must be admitted that the hope of improvement in the secondary class of cases is a small one.

### III.—CHRONIC.

Of 36 cases, 14 occurred in men and 22 in women. The average age of the men was 61·8 years and of the women 57·1 years. The youngest patient was 19 years old and the eldest

was 82. Arranged in decades, their age-distribution was as follows:—

Age.	Male.	Female.	Total.
20, or less, to 30 ... ..	0	1	1
31 to 40 ... ..	1	0	1
41 to 50 ... ..	1	5	6
51 to 60 ... ..	4	7	11
61 to 70 ... ..	5	5	10
71 to 80 ... ..	2	2	4
81 to 90, or more... ..	0	1	1
	13	21	34
Age not stated .. ..			2
Total ... ..			36

From this table it will be observed that 33 per cent. of the cases occur in persons between the ages of 51 and 60, and that 63 per cent. of the total number occur in patients of 51 to 70 years. A *good result* was obtained in eight instances. Brief particulars are tabulated below:—

Case.	Sex.	Age.	Duration.	Right Eye.		Left Eye.	
				Before.	After.	Before.	After.
44	F	53	3 years	6/6 with gl.	* 6/6 with gl.	6/5	6/6
45	F	52	5 years	*	6/9, J <sub>4</sub>	C/36	* 6/36, J <sub>4</sub>
46	F	59	2 years	*	6/9, J.		* 6/60 (Cataract)
47	M	64	Prophy- lactic		(H.M.)	6/60 (Cataract)	* 6/9
48	F	53	8 years	6/5 with gl	* 6/12, J <sub>2</sub>	6/60	* 6/12, J <sub>4</sub>
49	F	41	1½ years	6/12	6/18	6/12	* 6/9, J <sub>1</sub> .
50	M	37	10 months	P.L.	* 6/18	6/18 with gl.	P.L.
51	F	69	2 years	0	* 0	*	6/18

Five out of the eight cases recovered practically normal vision, and in every instance useful sight was retained. The prognosis clearly does not depend on the length of the history, for this is



often much longer than in the least successful cases. Neither, apparently, is the age of the patient a factor of prime importance, though none of the really aged patients appear in the list of conspicuous successes. The chief guide to the prognosis is the presence or absence of limitation of the field of vision, and of cupping of the optic disc. A slight narrowing of the whole field, or more commonly of the nasal side, is a sign of paramount importance. In the majority of the cases quoted above, which did well, this was either absent or present in such a mild degree that it disappeared after the performance of iridectomy. Cupping of the optic disc is a late sign, and means that the intra-ocular pressure has been increased for a considerable time. It is not to be regarded as an essential in the diagnosis: to do so would be to lose the only opportunity of doing any real good for the patient. The majority of cases of chronic glaucoma illustrate this point, not because surgeons have waited for cupping of the disc, but because the patients either have not sought advice until too late, or because they have refused early operation because they themselves have experienced no symptoms of the disease. Cupping of the disc, *per se*, is not harmful, it is simply a physical sign which indicates that the pathological process has been at work for a considerable time and has produced a condition which operation will not greatly benefit. It is the chronicity of the process that is its harmful feature; in this respect it rather recalls the effect of long-continued irritation in any other part of the body. Whereas a recent, severe irritation may end in complete recovery of function, fibrosis or even growth may follow a chronic process.

So also the intra-ocular tension may be very greatly raised for a short time (as in the acute cases) and but little harm result, whilst the occurrence of intermittent attacks of increase of pressure, even though they be moderate in degree, will so damage the structure and functions of the eye that recovery will be impossible. It frequently happens that patients present no signs of raised tension when seen by the oculist, though their history is clearly one of chronic glaucoma; yet all the time the process

is going on and causing the usual result of increased pressure anywhere in the body, namely, fibrosis. (This is really only a repetition of Thomson Henderson's theory; he maintains that sclerosis of the pectinate ligament is the essential factor, and that diminution of the filtering angle is only a secondary effect.) The result of the struggle between the surgeon and the fibrosis is the same in the eye as in other cases—fibrosis wins. In short, if we wish to do any good for patients with glaucoma, it is absolutely indispensable that we should diagnose the condition before secondary degenerative changes have taken place.

With regard to other signs of glaucoma, the *vision* varies enormously before operation. Sometimes it is 6/5, though the process is far advanced; in these cases the tension is not usually very high at the time of examination. Case 48 is an example of this kind; the series of perimeter charts given leaves no doubt as to the diagnosis, and also illustrates the recovery of the field after a timely iridectomy. In other instances the vision is considerably reduced, though a good result is eventually obtained. Frequently the sight continues to improve for some time after operation. This occurs in other cases as well, but is most marked in the "chronic" class. A few examples may be given:—

	Case.	Vision on Discharge.	Vision later on.	
CHRONIC ...	45	6/36 Tn	6/9, J <sub>4</sub> , T. +	2½ years
	49	6/24, 6/36	6/18, 6/9	3 weeks
	76	F. at 2ft 6/9	6/60, 6/6 and J <sub>2</sub>	3 years
PRIMARY ...	2	6/18 and J <sub>1</sub>	6/6 and J <sub>1</sub>	2½ years
	3	H.M. at 1ft	6/36	3 years
	5	4/60, Tn.	6/36	2½ years
SECONDARY	27	4/60	6/24, J <sub>16</sub>	2½ years
	40	No P.L.	F. at 7ft	1 month
			3/60	3 days later
			6/60	4 days later

None of the subjective symptoms are constantly observed. *Haloes* are the commonest, though very often absent. They may be due to mucous conjunctivitis or some other cause, and not necessarily to glaucoma. Thomson Henderson has pointed out that in glaucoma the red ring is outermost, and there is a band of darkness between the concentric rings and the light (Med. Ann., 1909). Pain occurs both as *photophobia* and as frontal *headache*. It is generally paroxysmal in character, and only one or two attacks may occur in a year or two.

When these symptoms persist or recur after operation it means that the tension is still up, and that the destructive process is still going on. Case 48 is an excellent example of this kind. After iridectomy the vision improved and the field increased; later on pain occurred occasionally in the eye and in the forehead, haloes re-appeared, and the change was reflected in the decrease of the field of vision (cf. the charts for 1909, 1910, and 1911). When the eye is also blind *excision* is indicated, and 16 patients in all had it performed for this reason; of these, 2 were acute cases, 7 were secondary, and 7 were chronic. This is equivalent to 13 per cent. of the acute cases, 25 per cent. of the secondary, and 19.5 per cent. of the chronic.

*Bad results* followed in 17 cases. In 13 of these the conditions were bad from the outset. Six of them had blind, painful eyes, and excision was performed as already mentioned (Cases 54, 55, 56, 57, 58, and 64). In No. 52 there was a two years' history, and the tension was +2, with a much contracted field. After operation the field failed to improve, and the vision was "fingers at 6 feet." There was early glaucoma in the other eye. This result is such as the chart of the field would lead one to expect. Repeated operations were performed in Nos. 59, 61, 62, and 64, in each case on account of persistence of symptoms after the first iridectomy. Vision was not improved in any case, whether sclerectomy or a second iridectomy was performed, though in three the pain was relieved.

In the remaining 4 cases, the vision was worse after operation. In Case 65 the sight had been failing for "a long time"; tension was +1, the disc was cupped, and  $V=6/18$  (some) with

glasses; there was no pain. After iridectomy, combined with sclerotomy, the iris prolapsed, and, later on, excision was necessary, the eye being painful and blind. In No. 66 vision was 6/9 before operation, but fell to nil after it, following prolapse of the iris and hæmorrhage. In No. 67 the reason for the disappointing result is not clear: the history was a short one (five weeks), and the fields were but little reduced; vision was 6/18 and J<sub>1</sub> with the right eye and 6/36 and J<sub>1</sub> with the left; after operation it fell to 6/60 and J<sub>14</sub>. There was never any pain, before or after. Vision was also reduced in Case 68 from 6/12 to 6/36 and J<sub>4</sub>, whilst the pain and coloured rings persisted. The original history extended over two years, and the case was remarkable in that the patient was myopic and only 19 years old. Herbert's operation was subsequently performed, and the tension fell satisfactorily, but the vision, at the time of writing, is unchanged.

The remaining 9 cases remained practically *in statu quo ante*, vision was not improved by operation, and the field was not affected, though in some instances pain and the other subjective symptoms were relieved. The important features of the cases are tabulated below:—

Case.	Sex.	Age.	Duration.	Right Eye.		Left Eye.	
				Before	After.	Before.	After.
69	F	—	1 year	Cannot read	* F. at 2 m.		Reads
70	F	48	1½ years	—	—	F. at 4ft *	F. at 4ft
71	M	62	—	—	6/9	P.L. *	H.M.
72	F	44	8 months	P.L.	P.L.	0 *	0
73	M	52	4 years	** Peripheral vision only (P.L.)			
74	F	58	10 years	6/12	* —	6/24 *	—
75	M	60	5 years		* <6/60, J <sub>κ</sub>		* 6/24, J <sub>14</sub> (Cataract)
76	F	65	4 months	<6/60	* F. at 2ft	6/9	6/6, J <sub>2</sub>
77	F	73	1 year	6/18	* 6/36, J <sub>4</sub>	6/24 *	6/36, J <sub>2</sub>
78	M	65	6 months	6/60	* 6/60	6/60	6/60
79	M	51	2 years	—	6/9, J <sub>4</sub>	*	6/24

*Cataract* occurred in 5 of the chronic cases, viz., in Nos. 57, 61, 73, 75, and 77; it was present in each eye before operation in all except No. 73, and in this instance, though it followed iridectomy, it was senile, and not traumatic in character. It was also noted, on admission, in Nos. 11 and 15 amongst the acute cases, and in Nos. 18, 20, 25, and 41 amongst the secondary cases.

#### CONCLUSIONS.

Whilst the primary acute cases give excellent results from iridectomy if seen within a month or so of the onset, it occasionally happens that a hæmorrhage occurs, and what should be a very successful operation is converted into a very mediocre one. This is the more regrettable, as the accident is one that it is difficult either to foresee or to avoid. Inasmuch as it occurs almost exclusively in patients with general arterio-sclerosis in whom the acute attack of glaucoma has probably been induced by some transitory rise of blood-pressure, it may be advisable in some cases to take measures for reducing blood-pressure and vascular congestion before operating. Saline purgation and local venæsection may be employed, whilst internally, iodides and vaso-dilator drugs such as erythrol tetranitrate may be taken. It is important that the patient should keep as quiet as possible in bed for the same reason. It has been suggested that pilocarpine drops should be used instead of eserine at the early part of the attack, because pilocarpine causes less vascular congestion. Massage of the eyeball has also been recommended strongly by those who have tried it, and, by analogy with the excellent results that are obtained in acute effusion into joints, might be expected to be very useful in promoting absorption of the exudate. It is not suggested that any of these methods should be employed as alternatives to iridectomy, but as antecedents to it. If assiduously carried out during the time that must elapse whilst preparations for operation are being made, they would probably effect a considerable improvement in the patient's condition and minimise the chance of a hæmorrhage taking place.

In any case, the time is not being wasted, for the cases quoted show that as long as the patient has iridectomy done fairly soon after the attack begins, the lapse of a few hours does not impair the prognosis to the extent that is commonly believed.

With regard to the secondary cases there is little to be said. The prognosis is bad, as a rule, and must remain so, unless more effective methods for dealing with the primary factor in the disease are introduced.

The striking feature about the chronic cases is that, whilst so many of them improve but little under treatment, one can never tell beforehand which will do well and which badly. In many instances the disease has gone irremediable lengths before the patient seeks advice. As with cancer, the onset is very insidious, and there is no prospect of teaching the public how to recognise its early stages. The gradual diminution of visual acuity, the occasional mistiness of the sight, the slight amount of pain, unassociated with any change in the external appearance of the eye, all these lead the layman to attribute the warnings of oncoming glaucoma to increasing years. It remains for the specialist to discover such cases at the earliest possible moment, for, as we have seen, the prognosis is still fairly good when degenerative changes in the eye have not begun. It is his duty to examine every person in whom experience has shown that there is a possibility of glaucoma by those means which reveal it most accurately. The field of vision should be carefully investigated with a perimeter and the fundus examined. The slightest narrowing of the field should be regarded with suspicion, and the patient advised to come at regular intervals for advice. Absence of pain, or of impairment of vision, must not influence the practitioner in the least—both are common in well-marked glaucoma. Bjerrum has advised the use of a special instrument, the Scotometer, for diagnosing the disease in an early stage. It is based upon the observation that "enlargement of the blind spot in the form of a narrow crescentic band of scotoma either above or below the central fixation point" is almost constant in early glaucoma. It is maintained that this test will give a positive

result at a time when the field of vision, taken in the ordinary way, is unaltered.

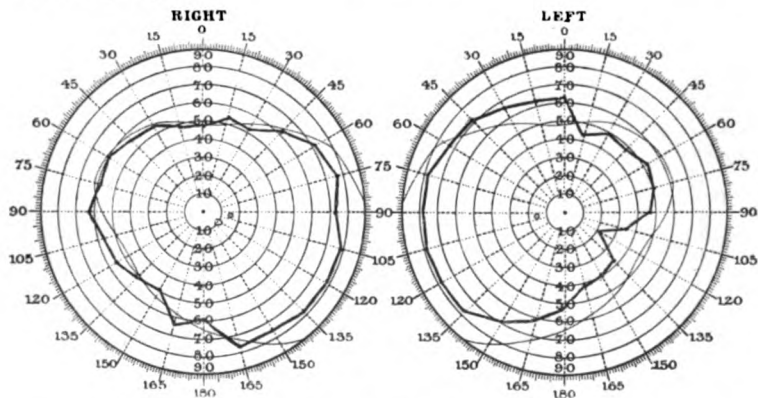
With regard to operative procedures, several methods have been suggested for the provision of satisfactory drainage from the eyeball in cases where iridectomy has failed. Herbert's operation is the latest development of the plan of making a leaking scar; time alone can show whether the result obtained is permanent. Up to now the tension has generally been reduced to a satisfactory extent, giving relief to the pain, though the vision has not been much improved; this, however, is not the fault of the operation. Knapp, and others, have recommended cyclodialysis in those patients in whom iridectomy has failed to relieve tension. Successful results have been reported in 11 out of 15 such cases, so that the method is at least worth a trial. A method that has never been applied in ophthalmic surgery, though it has given brilliant results elsewhere when drainage is obstructed, is lymphangioplasty. True, the conditions in the eye are different from those existing in any other part of the body; it is impossible to render the conjunctival sac aseptic. Yet the eye tolerates a foreign body introduced into it with ordinary antiseptic precautions, as in Mules' operation. It does not become infected through the ever open door resulting from a "leaking scar" operation. There seems to be no a priori reason why a silk thread, skilfully introduced through a subconjunctival wound should not remain in place for years and maintain a satisfactory drainage from the interior of the eye. At any rate, the method seems worthy of trial in those cases where it is obvious that sight will be entirely lost unless something effective is done, and that quickly.

## ACUTE.

Case 1.—85, E., 1908.—G. H., male, aged 56. Admitted March 29th, discharged April 8th. C.O.A.: Cornea, right eye, hazy and anæsthetic; tension, right eye,  $+0.0$ .D. cupped, left eye,  $+0.0$ .D. much cupped; vision, right eye, no P.L. Treatment: March 29th, right eye, iridectomy; April 5th, right eye, with  $+1=6/12$ , field normal. C.O.D.: Vision, April 22nd, right eye, with  $+2$  sph. and  $+2$  cyl.  $120^{\circ}=6/6$ .

Present condition (April 11th, 1911): Vision, without glasses, right eye, 6/9, left eye, 6/6 (some); with glasses, both eyes,  $J_1$  with  $+2.75$ .

Case 2.—171, E., 1909.—E. S., female, aged 56. Admitted August 3rd, discharged August 16th. History: Sudden blurring of vision of left eye; duration of attack, six days; headache and ocular pain; subjective symptoms, coloured rings, no vomiting. C.O.A.: Cornea, left eye, hazy, shallow anterior chamber; tension, left eye,  $+3$ ; fields, both eyes, full; vision, left eye, H.M. Treatment: August 3rd, left eye, iridectomy. Progress: T—, but vision impaired. C.O.D.: Vision, right eye, 6/9 with  $+1.5$  cyl.  $120^{\circ}$  and  $J_1$  with  $+5$  sph.; left eye, 6/18 with  $+1$  cyl.  $150^{\circ}$  and  $J_4$  with  $+3$  sph.

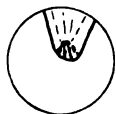


Present condition (March 25th, 1911): Left eye, good coloboma, no pigment in scar, thin, bluish-grey sclerotic, old scleritis; tension, both eyes, normal; fields, vide charts (full); vision, without glasses, right eye, 6/18, left eye, 6/12 (2); with glasses, right eye, 6/6 (4) with  $+2$  sph. and  $+1$  cyl., and  $J_1$  with  $+4.75$  sph., left eye, 6/6 with  $+1.25$  cyl.  $150^{\circ}$  and  $J_1$  with  $+2.75$  sph. and  $+1.5$  cyl.  $150^{\circ}$ .

Case 3.—86, Or., 1908.—F. W., female, aged 65. Admitted May 4th, discharged May 16th. History: Influenza three weeks ago; sudden intense blinding pain in left eye; right eye also bad, but got better. G.P.

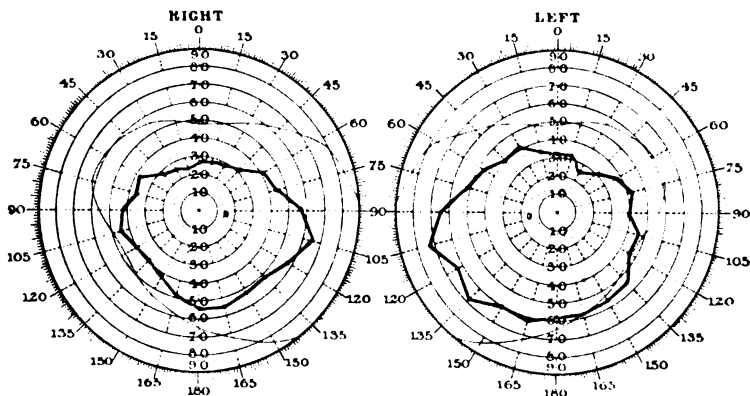


instilled atropine into right eye. C.O.A.: Both eyes, lens opacities; tension, right eye,  $+2$ ; fields, right eye, very much contracted; left eye, contracted; vision, right eye, shadows at 1 ft.; left eye, 6/24 (2). Treatment: May 5th, right eye, iridectomy; left eye, eserine.



Present condition (March 24th, 1911): Right eye, good coloboma, but opacities (see figure); left eye, shallow anterior chamber, hardly any reflex; lens opacities; tension, N; vision, without glasses, both eyes, 6/36; with glasses, can read and sew. No pain or any symptoms of T $+$  in either eye.

Case 4.—153, E., 1908.—D. P., female, aged 57. Admitted November 9th. History: Duration of attack, three weeks; acute pain, blurring and failure of sight, left eye; subjective symptoms, vomited all day. C.O.A.: Cornea, left eye, slightly hazy; tension, right eye, N., left eye,  $+2$ ; fields, right eye, N., left eye, some nasal contraction. Treatment: November 9th, left eye, iridectomy upwards. C.O.D.: Tension, left eye, N; vision, right eye, 6/9; left eye, 3/60, no cupping of O.D.



Present condition (March 14th, 1911): Tension, both eyes, N; fields, wide charts; vision, with glasses, right eye, 6/6 (full) with  $+2$  sph. and J $_1$  with  $+5$  sph.; left eye, 6/9 with  $+1.5$  cyl. 150° and J $_1$  with  $+2$  sph. and  $+1.5$  cyl. 150°. No floating bodies; no rings; occasional shooting pains in left eye; had supra-orbital and nasal neuralgia in right eye two months ago; iridectomy, right eye, advised; patient refused.

Case 5.—11, E., 1908.—J. B., female, aged 53. Admitted December 4th, discharged January 5th. History: Sudden pain in right eye ten

weeks ago; pain made worse by drops put in at the infirmary; vomiting. C.O.A.: Cornea, right eye, hazy; iris and pupil, fixed, dilated, clear; tension, +. Treatment: December 5th, right eye, iridectomy; December 13th, left eye, acute glaucoma; iridectomy. Progress: December 31st, right eye, small cystoid scar. C.O.D.: Tension, right eye, N.; vision, right eye, 4/60, field greatly contracted; left eye, 6/60, field nearly normal.

Present condition (May 21st, 1911) from O.P. letter: Left eye, field unchanged; vision, without glasses, right eye, 6/60, left eye, 6/36; with glasses, right eye, 6/36, left eye, 6/18.

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Case 6.—238, Or., 1908.—H. G., female, aged 63. Admitted December 9th, discharged December 23rd. History: "Bilious attack" with failure of vision a fortnight ago; subjective symptoms, several attacks of vomiting. C.O.A.: Cornea, left eye, hazy; iris and pupil, dilated and fixed; tension, +3. Treatment: December 15th, left eye, iridectomy, after treatment by eserine. Progress: December 18, left eye, wound leaking; much lens opacity.

Present condition (March 27th, 1911) by letter: Distant and near vision good in each eye. No pain, headaches, coloured rings, or blurring of vision at any time.

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Case 7.—40, E., 1908.—E. C., male, aged —. Admitted May 27th, discharged June 4th. History: Haloes seen round the lights at Christmas time; subjective symptoms, dizziness. C.O.A.: Tension, left eye, +; field much contracted; vision, 6/36 and J<sub>2</sub> with glasses. Treatment: June 5th, left eye, iridectomy. C.O.D.: Vision, left eye, 6/12 with +1.5 cyl., J<sub>2</sub> with +3.5 sph. and +1.5 cyl. Field unchanged.

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Case 8.—159, Or., 1909.—H. G., male, aged 55. Admitted August 28th, discharged September 11th. History: Acute glaucoma on day of admission. C.O.A.: Tension, left eye, +2; field, O.D. normal; vision, right eye, print at 10 inches. Treatment: August 28th, left eye, iridectomy. C.O.D.: Vision, left eye, 6/18 (2), slight corneal haze.

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Case 9.—E.—L. H., female, aged 49. History: Acute glaucoma, left eye, 1890.

Present condition (March 14th, 1911) Left eye, small nebula, central lens opacity; a.c. shallow above; tension, right eye, +1. left eye, N.; fields, right eye, O.D. and field normal, left eye, O.D. pale with scleral ring; vision, without glasses, right eye, 6/36; with glasses, right eye, 6/9 with +2.75 sph. and J<sub>1</sub> with +7.0 sph.; left eye, fingers at 5 feet.

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Case 10.—169, Or., 1908.—M. A. K., female, aged 74. Admitted September 16th, discharged September 23rd. History: Treated by Mr. Higgins, 1904. Patient could see to read until a fortnight ago; pain.

C.O.A.: Cornea, right eye, steamy, with vesicle on inner side; iris and pupil, right eye, dilated and fixed; tension, both eyes, +2; field, right eye, nasal contraction; vision, right eye, fingers at 1 foot. Treatment: September 16th, right eye, iridectomy with sclerectomy. C.O.D.: Vision, right eye, fingers at 1 foot.

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Case 11.—35, E., 1909.—C. C., female, aged 62. Admitted August 16th. History: Duration of attack, five weeks; two or three attacks of pain; subjective symptoms, headache and nausea. C.O.A.: Cornea, left eye, steamy and anæsthetic, with episcleral injection; exophthalmos: iris and pupil, large, oval, and non-reacting; vision (myopic) reduced to a minimum. Treatment: Left eye, iridectomy. C.O.D.: Left eye, can see some figures on a watch.

Present condition (March 27th, 1911): Iris and pupil, right eye, circular, react well; left eye, no reaction, pieces of iris floating in the pupil, lymph on cornea, also deep in a.c.; lens amber and opaque: tension, right eye, N., O.D., normal, reddish; fields, right eye, early central opacity of lens; left eye, good projection of light, but hardly any fundus reflex; vision, with glasses, right eye, 6/12 (3) and J<sub>2</sub>; left eye, fingers at 1 foot. Right eye, yellow and silvery sparks around lights: left eye, occasional slight pain.

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Case 12.—39, E., 1908.—L. C., female, aged 57. Admitted March 24th and April 28th, discharged April 14th and May 28th, respectively. History: Sight was bad eighteen months ago; then patient had an attack of influenza in August; the eyes suddenly became bad again, and the sight grew steadily worse. Latterly the decline of vision has been rapid. C.O.A.: Cornea, both eyes, hazy, anæsthetic; right eye, iris atrophic, pupil dilated and fixed, lens rather hazy; left eye, pupil dilated and fixed; tension, right eye, +3; fields, both eyes, much contracted, O.D. much cupped, and pale in outer half; vision, right eye, 6/18, left eye, P.L. Treatment: March 26th, right eye, iridectomy; March 30th, T+, lens capsule found wounded. March 30th, left eye, acute glaucoma; March 31st, left eye, iridectomy. April 28th, right eye, pain: T+2; cornea steamy; iris pigment in wound; nasal limitation of field: shallow a.c.; vision=P.L.; iridectomy and sclerotomy. May 4th, bulging scar with iris in it; opacity of lower half of right cornea. May 18th, cyclodialysis. April 28th, tension, left eye, T+. C.O.D.: Not much improved.

Present condition (March 14th, 1911) by letter: P.L. only in each eye.

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Case 13.—66, E., 1909.—C. E., female, aged 57. Admitted February 6th, discharged March 31st. History: Left eye, misty and right eye healthy two years ago; right eye misty for past two months. C.O.A.: Cornea, right eye, hazy, shallow a.c.; pupil, dilated; tension, +3; fields, right eye, O.D. cupped and atrophic; left eye, full. Treatment: February 6th, right eye, iridectomy; February 25th, left eye, iridectomy. C.O.D.: Tension, right eye, N; vision, right eye, no P.L.; left eye, 3/60.

Case 14.—108, E., 1906.—A. L., female, aged 64. Admitted January 10th, discharged February 5th. History: Has worn glasses for years: sight of left eye began to deteriorate a year ago; in September it began to be much worse; hæmorrhagic retinitis diagnosed at O.-P.: severe pain at times; acute glaucoma in January, 1907, after an illness. C.O.A.: Cornea, left eye, hazy; iris and pupil, left eye, dull, irregular, and fixed; tension, left eye, +2, fundus not seen; vision (November, 1906). right eye, 6/9 with +3 sph.; left eye, fingers at 5 ft., with +2 sph.: on admission no P.L. Treatment: January 10th, left eye, iridectomy. Progress: Left eye, pain increased. C.O.D.: Tension, right eye. N.: left eye, excision.

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Case 15.—197, E., 1909.—E. W., female, aged 63. History: Increasing loss of sight and frontal pain in right eye for one month. C.O.A.: Both eyes, lens sclerosis; tension, right eye. T—: vision, right eye. no P.L.: left eye, 6/5 with +3.75 sph. Treatment: March 6th, right eye, iridectomy. Progress: Pain; wound bulging; extrusion of vitreous: no P.L. C.O.D.: Right eye, T+2. Excision.

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## SECONDARY.

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Case 16.—66, Or., 1906.—J. D., male, aged 60. Admitted June 14th, discharged July 10th. History: Corneal ulcer, left eye, perforated, cautery, three weeks ago; glaucoma developed; much pain. C.O.A.: Cornea, right eye, clear; left eye, anterior staphyloma, and scarring, no a.c.; iris and pupil, right eye, N.; tension, left eye, +2—3. Treatment: June 15th, left eye, iridectomy. C.O.D.: Tension, left eye, T+1, lens opaque.

Present condition (May 12th, 1911): Iris and pupil, right eye, N.; tension. N.; field, full; vision, without glasses, 6/9, with glasses, not improved. No pain, rings, or signs of + tension; never red. Left eye blind; no more pain.

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Case 17.—116, 1907.—R. M., male. Admitted November 22nd, discharged December 4th. History: Under Mr. Brailey, 1900 (Feb. Rep. 30) "Injury seven months ago; gradual failure of vision; right eye, 6/36, T+; left eye, no P.L., T+1; iridectomy on right eye; left eye, dislocated lens; discoloured iris." C.O.A.: Cornea, right eye, N; left eye, infected and infiltrated, blood and pus in a.c.; right eye, coloboma, ? post. synechiæ; left eye, pupil (with eserine), dilated and discoloured, no reaction; tension, right eye, Tn, left eye, T+2; fields, right eye, lens opacity, disc pale and slightly cupped; left eye, fundus not seen; vision, left eye, no P.L. Treatment: November 25th, left eye, excision after failure of attempt at iridectomy. Progress: December 3rd, V=less than 6/60, disc greyish, shallow cupping. C.O.D.: Ring of choroidal atrophy round disc. Vision, less than 6/60. Left socket healthy.

Case 18.—28, 1906.—T. B., male. Admitted June 23rd. History: Grit in right eye three months ago; the eye became inflamed; the pain went away after bathing with hot lotion, but the eye remained red, and the vision was bad. C.O.A.: Staphyloma at each angle. hyphema, opacities; iris discoloured; tension, T+1; vision, no P.L. Treatment: Excision; no growth found.

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Case 19.—166, 1908.—J. R., male, aged 26. Admitted June 20th and 24th, discharged July 2nd and 6th respectively. History: Right eye, needling fourteen years ago. Right eye much inflamed, after coal dust in it. a fortnight ago. C.O.A.: Right eye, cornea abraded and hazy, small hypopyon, tremulous iris, deep a.c., lens opaque, capsule at outer side of pupil; tension, right eye, T+1; left eye, opaque nerve fibres under fundus: vision, right eye, P.L.; left eye, 6/18. Treatment: June 25th, right eye, hypopyon gone; sclerotomy. Progress: Eye still painful. C.O.D.: Enucleation.

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Case 20.—14, 1907.—F. B., female, aged 56 years. Admitted February 20th. History: Previous vision, November, 1906, cataract extraction, right eye. A few weeks ago right eye became very painful; subjective symptoms, vomiting. C.O.A.: A.c., old and recent blood; cornea, hazy, no k.p.; iris and pupil, right eye, discoloured, coloboma, some capsule; left eye, commencing cataract; tension, right eye, +2, left eye, N; field, right eye, fundus not visible; vision, right eye, slight P.L.; left eye, counts fingers, can see distant letters. Treatment: February 21st, paracentesis, colour of iris and haziness of cornea at once improved. Progress: February 25th, T+3; February 28th, T+2; March 14th, downward iridectomy. C.O.D.: Tn; no pain.

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Case 21.—98, 1907.—H. J., female, aged 74. Admitted March 4th. History: Failing sight in each eye for three years; present condition, two months. C.O.A.: Both corneæ hazy; iris and pupil fixed, both lenses dislocated forwards, partially opaque; tension, both eyes, T+1; vision, right eye, P.L., left eye, no P.L. Treatment: March 5th, iridectomy and extraction of right eye. March 12th, cornea sloughed; panophthalmitis. C.O.D.: Right eye, no P.L.

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Case 22.—62, E., 1908.—J. E., female, aged 43. Admitted March, discharged April 1st. History: Right eye blind and painful, and has been divergent for nine years. C.O.A.: Cornea, hazy; iris and pupil, fixed, no reflex; tension, +1; lens floating in vitreous; vision, right eye, no P.L.; left eye, less than 6/60, and 6/18 (6) with -4.0 sph. and J<sub>1</sub> at 8 inches. Treatment: March 25th, right eye excised.

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Case 23.—39, Or., 1908.—J. G., male, aged 65. Admitted March 5th, discharged April 3rd. History: Seven days' pain in left eye. C.O.A.:

Cornea, opaque at upper edge, shallow a.c.; lens in a.c., blood behind it. Treatment: March 5th, extraction of lens, extrusion of vitreous, some soft material left. Progress: March 31st, iritis all cleared up, cornea hazy, ulcer galvano-cauterized. C.O.D.: Vision, right eye, 6/12 (some). left eye, V=P.L.

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Case 24.—78, Or., 1908.—J. M., male, aged 63. Admitted April 22nd, discharged April 29th. History: Blow, left eye, nine weeks ago; much pain. C.O.A.: Iris and pupil fixed and dilated, lens in a.c., opaque; tension, +2. Treatment: Excision.

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Case 25.—79, 1906.—H. G., male, aged 32. Admitted November 15th, discharged December 4th. History: Rheumatic fever three times. C.O.A.: Cornea, both eyes, clear, no k.p.; right eye, shallow a.c.; iris bombé, occluded; left eye, white lines in iris, reacts; tension, both eyes, Tn; field, left eye, faint lens opacity; vision, right eye, ? P.L. Treatment: November 22nd, right eye, iridectomy. Progress: December 3rd, right eye, no P.L., calcareous lens capsule.

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Case 26.—103, 1908.—G. J., male, aged 58. Admitted September 12th and November 7th, and discharged October 7th and November 20th, respectively. History: Acute rheumatism, recurrent iritis (first in 1881); four years ago left eye affected, now quite blind; recently right eye painful and sight poor. C.O.A.: Left eye, no anterior chamber; right eye, pupil very small, lymph; left eye, pupil very small, organised lymph, iris muddy; tension, right eye, ?+; left eye, +2; left eye, no fundus reflex; vision, right eye, 6/36, left eye, P.L. (with good light). Treatment: September 14th, right eye, iridectomy. C.O.D.: October 5th, vision, right eye, 3/60; November 9th, Tn, V=6/60, field almost normal. October 5th, left eye, quite blind; November 9th, T+2, V=0; iridectomy. C.O.D.: Sees H.M., Tn.

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Case 27.—167, E., 1909.—T. R., male, aged 61. Admitted January 21st, discharged February 3rd. History: Sensation of foreign body in the eye; rheumatic history; denies syphilis and gonorrhœa; sharp pain in right eye a fortnight ago; total loss of vision in right eye. C.O.A.: Right eye, hyphæma; iris bombé; tension, N. Treatment: January 21st, iridectomy. C.O.D.: Vision, 4/60.

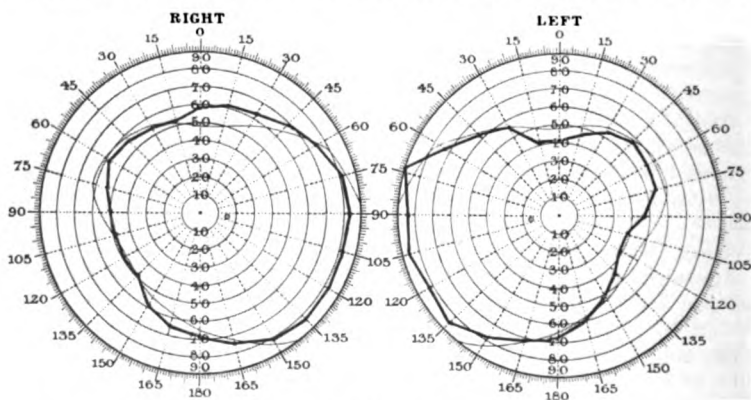
Present condition (March 28th, 1911): Right eye, good, clear coloboma; left eye, iris circular, reacts well: a.c. N, pinguecula; tension, both eyes, N; fields, right eye, some general contraction; left eye, full; vision, right eye, without glasses, fingers at 4 ft., left eye, 6/6; with glasses, 6/24 with -4 cyl. 40° and J<sub>16</sub> with +3 sph. and +1 cyl. 130°; left eye, J<sub>1</sub> with +3 sph. Occasional coloured rings right eye, and sometimes mistiness for a few seconds, both eyes. Vision of right eye much less acute on some days than on others. Patient always cures this with a "dose of salts."

Case 28.—125, E., 1906.—S. N., female, aged 68. Admitted September 18th, discharged October 20th. History: Has had rheumatic fever; recurrent attacks of pain and diplopia. C.O.A.: A.c., both eyes, N.; cornea, both eyes, N; iris and pupil, right eye, N; left eye, pupil very small; iris reacts; tension, right eye, N; left eye, +, slight lens opacity; vision, right eye, 6/9 with +1 sph. and +1.5 cyl.; left eye, fingers at 1 ft. Treatment: September 20th, left eye, upward iridectomy. C.O.D.: Vision, left eye, fingers at 5 ft.



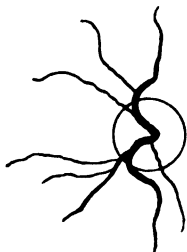
Present condition (May, 1911): Left eye, iris drawn up, occluding pupil, dense broad scar (see figure); tension, both eyes, N; field, right eye, almost reduced to fixation point; vision, without glasses, 6/18. left eye, blind. Patient sees black circles when looking at anything at twilight; never any coloured lights. Right eye never painful; left eye occasionally painful.

Case 29.—44, 1908.—S. C., male, aged 46. Admitted January 1st, discharged January 23rd. History: Three previous attacks of iritis (syphilis 16 years ago, treated with mixture for 4-6 months). Fifteen days ago another attack with neuralgia and mistiness. C.O.A.: Cornea, right eye, hazy, left eye, clear; iris and pupil, right eye, browner than left, injected, poor reaction, posterior synechia, organised lymph in pupil; left eye, iris clear, posterior synechia; tension, right eye, +, left eye, N; vision, right eye, hand movements. Treatment: January 13th, right eye, iridectomy, upward. C.O.D.: Could see to read a newspaper with glasses.



Present condition (March 17th, 1911): Right eye, a little lymph around pupil edge, total posterior synechia, coloboma, clear; left eye, iris reacts, small, regular; tension, right eye, +; O.D. N, scleral ring, hazy media;

left eye, N (?—), O.D. ditto; fields, both eyes, full (vide charts); vision, without glasses, right eye, 6/36, left eye, 6/18; with glasses, right eye, 6/9



with  $+1.75$  sph. and  $+0.75$  cyl and J<sub>2</sub> with  $+4$  and  $+0.75$ , no pain, no coloured rings; left eye, 6/6 with  $+1.75$  sph. and J<sub>2</sub> with  $+3.5$  sph., no symptoms.

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Case 30.—19, 1908.—L. B., female, aged 19. Admitted October 28th and November 21st, and discharged November 7th and January 6th respectively. History: Perfect sight till eleven years ago; I.K.; four years ago operation each eye; hæmorrhage each eye; right eye quite blind; left eye failing eighteen months. C.O.A.: Cornea, right eye, small and calcareous, left eye, some sclerosis and opacities; iris and pupil, right eye, occluded, left eye, optical iridectomy, down and in; tension, right eye, 0; left eye,  $+$ ; both eyes, nystagmus; left disc pale, choroiditis; vision, right eye, 0, left eye, P.L., no projection. Treatment: Nine Soamin injections. Acute arsenical poisoning.

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Case 31.—58, E., 1909.—B. D., female, aged 32. Admitted December 29th, discharged February 23rd. History: Iridocyclitis, k.p.; pain in right eye four months ago and again later on; no history of syphilis or tuberculosis. C.O.A.: Cornea, right eye, hazy, k.p.; iris and pupil, muddy, dilated, small hypopyon; tension,  $+$ , some deposits on lens; vision, H.M. 2 ft. Treatment: Atropine and inunc. hydrarg. C.O.D.: Tension, right eye,  $+$ ; vision, right eye, 6/60, still much k.p.; left eye, 6/5.

Present condition (June 19th, 1911): Iris and pupil, right eye, round, no synechiae, no k.p., rather sluggish reaction to light; right eye, O.D. normal, T.  $+$ ; left eye, N; fields, both eyes, full; vision, without glasses, right eye, 6/6 (6), left eye, 6/5. Occasional mild frontal headaches and pain in right eye; no further iritis; no glaucomatous symptoms. Treatment in ward was mercury by baths and inunctions; all the left lower teeth have come out; and three sequestrotomies for mercurial necrosis have been performed elsewhere. At Croydon General Hospital the original iridocyclitis was regarded as tuberculous in nature. No tuberculous lesions have since appeared.

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Case 32.—117, E., 1909.—M. K., female, aged 26. Admitted September 26th, discharged October 16th. History: Iritis four years ago for a week,



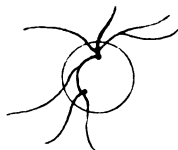
again two years later and during last five months. C.O.A.: Left eye, atropine dermatitis; iris and pupil, left eye, blurred, small, posterior synechiæ; tension, right eye, N, left eye,  $\frac{+}{-}$ . Treatment: September 29th, left eye, iridectomy.

Case 33.—130, E., 1909.—H. M., male, aged 36. Admitted February 22nd, discharged March 5th. History: Twenty-one years previously, left eye blinded by injury; five weeks ago it became painful. C.O.A.: Cornea, left eye, opaque, vascularised, very shallow a.c.; pupil, left eye, occluded, iris bombé; tension, right eye, N; left eye,  $\frac{+}{-}$ 1; vision, right eye, 6/9. Treatment: February 27th, left eye, excision.

Case 34.—126, 1907.—J. M., female, aged 50. Admitted January 24th and April 17th, and discharged February 13th and May 8th, respectively. History: Sight began to be troublesome last spring: sharp attack of pain December 6th, 1906; subjective symptoms, rings round gas lamps. C.O.A.: Cornea, both eyes, hazy; iris and pupil, both eyes, fixed and rather oval; tension, right eye,  $\frac{+}{-}$ 3 or 4, cupped disc; left eye,  $\frac{+}{-}$ 1, disc pale and much cupped; fields, right eye, contracted; left eye, contracted, central scotoma; vision, right eye, 6/36; left eye, 6/60, movements at 2 yards. Treatment: January 24th, right eye, iridectomy. Re-admitted April 17th, pigment in wound and on lens capsule. Vision, right eye, with  $\frac{+}{-}$ 5 sph. and  $\frac{-}{-}$ 5 cyl.=6/12 (2); with  $\frac{+}{-}$ 3.5 sph. and  $\frac{-}{-}$ 5.5 cyl.=J<sub>1</sub>; left eye, T $\frac{+}{-}$ 2, V=hand movements. Iridectomy, old iritis, April 29th. C.O.D.: Fingers at lower part of field.

Present condition: Coloured rings and flashes of light; distant vision bad; can see to read at about 14ins. "Left eye of no use to me." Occasional slight pain, never severely inflamed.

Case 35.—43, Or., 1906.—D. C., male, aged 46. Admitted April 9th, discharged May 16th. History: Failure of sight in left eye; duration of attack, nine months; several attacks of iritis. C.O.A.: Iris and pupil,



left eye, irregular and badly reacting; tension, right eye, N, left eye,  $\frac{+}{-}$ . O.D., much cupped; field, right eye, full; fundus of left eye as indicated; vision, right eye, 6/9, H.M.  $\frac{+}{-}$ 1 sph., left eye, P.L. Treatment: April 21st, left eye, iridectomy.

Case 36.—141, 1908.—A. A. M., female, aged 72. Admitted May 14th, discharged June 9th. History: Two previous attacks, one of them over a year ago; duration of attack, one month. C.O.A.: Iris, both

eyes, dilated and fixed; tension, right eye, +3; left eye, +2. Treatment: May 14th, right eye, iridectomy; left eye, eserine. Progress: right eye, V=3/6; May 25th, left eye, iridectomy. C.O.D.: Vision, right eye, 6/60; left eye, painful.

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Case 37.—217, Or., 1908.—M. A., female, aged 61. Admitted November 12th, discharged December 2nd. History: Left eye weak for several years; eye inflamed and sight failing, three weeks; constant watering. C.O.A.: Cornea, left eye, steamy, k.p. on back; iris and pupil, small fixed, discoloured; tension, +1; vision, right eye 6/24 (1); left eye, fingers at 2 yds. C.O.D.: Left eye, pupil dilates fully and in circular shape, with atropine.

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Case 38.—77, E., 1909.—S. F., female, aged 67. Admitted July 10th, discharged October 12th. History: 1906, cataract extraction, right eye; 1909, iridocyclitis and glaucoma. C.O.A.: Right eye, coloboma, anterior synechiae, aphakia, web; tension, +; vision, 6/18 after division of web; left eye, 6/18. Treatment: August 5th, right eye, paracentesis. Progress: November, re-admitted with pain and T+3, anæsthetic cornea, muddy iris, etc.

Present condition (May, 1911): Left eye, with glasses, 6/12 with +4.0 sph. and J<sub>12</sub> with +7.5 sph. A little pain occasionally; no coloured rings round lights.

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Case 39.—111, 1907.—J. L., male, aged 50. Admitted February 9th. History: Right eye becoming blind three years. C.O.A.: Cornea, right eye, hazy, deep a.c.; iris and pupil, small, muddy, sluggish, lymph; left eye, oval and sluggish; tension, right eye, +2; left eye, N; vision, right eye, none; left eye, 6/12 (2). Treatment: February 11th, iridectomy, vitreous escape, hæmorrhage into a.c., much pain. Tension equal in both eyes. Progress: March 9th, P.L. only, and still painful. Excision.

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Case 40.—101, 1907.—J. K., male, aged 76. Admitted October 25th and November 27th, and discharged November 13th and December 21st, respectively. History: Considerable pain in right eye; left eye infected and painful. C.O.A.: Left eye, old iritis, some lens opacities, pupil large and inactive; tension, right eye, +2; left eye, normal; field, left eye, full; vision, right eye, no P.L.; left eye, some pain, fair distant vision, but unable to read. Treatment: October 28th, right eye, iridectomy. C.O.D.: Right eye, no P.L.; left eye, iridectomy. C.O.A. (second): Right eye, pain, no P.L., good coloboma; December 2nd, V=fingers at 7 ft.; 5th, V=3/60; 9th, V=6/60; left eye, pupil small, with adhesions, under eserine, no pain; V=fingers at 4 ft.; Tn. C.O.D. (second): December 19th, no cupping of either disc; left eye, V=6/60 and J<sub>4</sub>, better with -6.0 sph.

Present condition (March 21st, 1911): Right eye, no anterior chamber, lymph around edges of coloboma, amber lens opacity, no P.L., sectors well seen by oblique illuminator, some opacity of Descemet's membrane, total posterior synechiæ, little fundus reflex; left eye, iris does not react, shallow a.c., some diffuse lens opacity (central); a little lymph at edge of coloboma; neither disc visible; vision, right eye, 6/60; left eye, 6/36 with  $-5$  sph. (prefers this to cylinder),  $J_{12}$  without glasses. No pain; always coloured rings round lights at night; no floating bodies; no further iritis.

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Case 41.—90, 1907.—W. H., male, aged 46. Admitted January 8th, discharged February 13th. History: Vision gradually failing for some years; unable, during last three weeks, to do tailoring. C.O.A.: A.c., right eye, very shallow: cornea, right eye, N; iris and pupil, right eye, small, irregular, excentric, iris cloudy, dull, bombé, no reflexes; left eye, pupil central, reflexes present, right lower part of lens opaque, rest somewhat opaque; tension, right eye,  $+$ ; left eye, Tn; vision, right eye, totally blind; left eye, fingers at 2 ft., good P.L. Treatment: January 14th, right eye, upward iridectomy. January 21st, hyphæma, bead of vitreous. April 8th, left eye, lens opaque, found to be dislocated upwards and outwards, dense opacities; no operation.

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Case 42.—7, E., 1909.—A. A., female, aged 31. Admitted May 19th, discharged May 28th. History: Recurrent iritis fifteen years; dense leucomata in both eyes at lower and outer parts of cornea; staphylomata. C.O.A.: Cornea, both eyes, leucomata; right eye, iris adherent to lens capsule, no reaction; left eye, iris attached to cornea below; tension, right eye, normal; a.c. shallow; left eye, T $+$ , a.c. very shallow; vision, right eye, no P.L., left eye, H.M. Treatment: May 21st, both eyes, iridectomy.

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Case 43.—141, E., 1909.—E. M., female, aged 51. Admitted April 28th and July 21st, discharged May 8th and July 31st, respectively. History: Pain and inflammation of eyes thirty years; iritis treated by atropine. C.O.A.: Both eyes, small nebulae; left eye, shallow anterior chamber; iris and pupil, both eyes, irregular, posterior synechiæ; tension, left eye,  $+$ ; vision, right eye, 6/9, with glasses; left eye, 6/36. Treatment: April 29th, left eye, iridectomy. July 25th, right eye, iridectomy. C.O.D.: Tension, right eye, Tn.

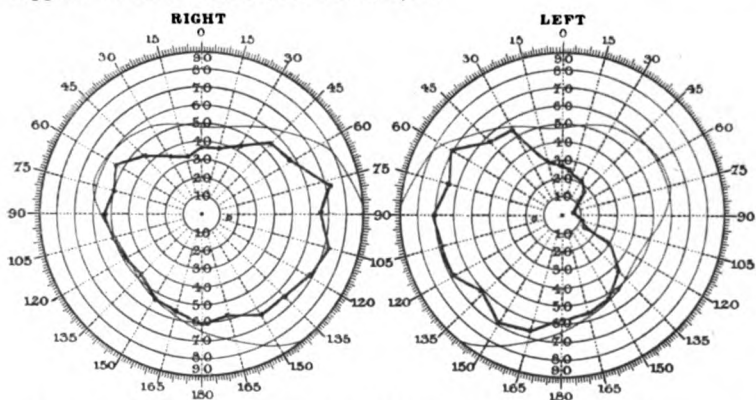
Present condition (March 24th, 1911): Sight of right eye seemed to fail rapidly since operations; without glasses, cannot see across street, slight pain, of shooting nature, "if doing too much." No pain in left eye. April 25th, corneal opacities right eye. Glasses ordered, right eye,  $+3$  sph. and  $+3$  cyl.; left eye,  $+4$  sph.; constant wear.

## CHRONIC.

Case 44.—129, E., 1909.—M. M., female, aged 53. Admitted April 4th, discharged May 4th. History: Rings, right eye, three years. C.O.A.: A.c., right eye, shallow; left eye, N; iris and pupil, both eyes, fixed and dilated; O.D. normal; vision, right eye, 6/9 and 6/6 with  $+1.0$ ; left eye, 6/5. Treatment: Right eye, iridectomy. C.O.D.: Fields a little contracted; vision, 6/6 (2) with  $+1.0$ .

Present condition (March 28th, 1911): Right eye, good clear coloboma; left eye, very well under eserine; tension, right eye, normal; O.D. not cupped, arterio-sclerosis; left eye, normal; vision, without glasses, right eye, 6/6. J<sub>1</sub> with  $+3.5$ ; left eye, 6/6; with glasses, right eye, 6/6 with  $+1.0$  sph. No pain; slight coloured rings.

Case 45.—102, 1908.—E. J., female, aged 52. Admitted September 14th, discharged September 26th. History: Has always worn glasses; iridectomy, left eye, five years ago (Mr. Brailey); left eye troublesome for a year; pain, floating bodies, failing sight, and spots for past six months. Treatment: September 15th, right eye, iridectomy. Progress: O.D. not cupped. C.O.D.: Tension, N; vision, 6/36.



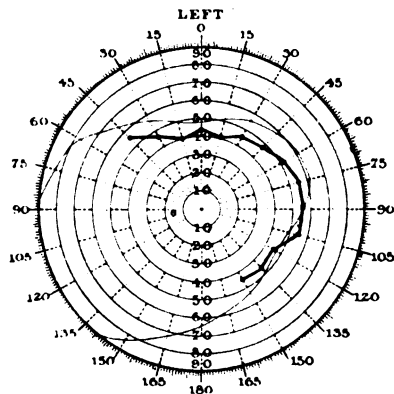
Present condition (March 14th, 1911): Right eye, wide coloboma; left eye, coloboma; tension, right eye,  $+$ ; O.D. reddish, no cupping; left eye, Tn, O.D. pale, vessels bent, lamina cribrosa well seen; fields, right eye, full; left eye, no nasal field (see charts); vision, with glasses, right eye, 6/9, with  $+3$  sph. and  $+0.75$  cyl.  $90^\circ$ , and J<sub>4</sub> with  $+5$  sph. and  $+1$  cyl.; left eye, 6/36 (1) with  $+3$  sph.  $+1.25$  cyl. and J<sub>4</sub> with  $+5$  sph. and  $+1$  cyl.

Case 46.—30, 1909.—S. A. C., female, aged 59. Admitted September 24th. History: Glasses, six years; two years ago, attack of pain, photophobia and dimness of vision, right eye, three others subsequently. C.O.A.: A.c., right eye, shallow; cornea, right eye, steamy; left eye, a.c.

and cornea normal; iris and pupil, right eye, discoloured, large, oval, fixed; left eye, discoloured, small (eserine), oval; tension, right eye, +; left eye, N; field, right eye, greenish reflex. Treatment: September 24th, right eye, iridectomy; September 28th, Tn. October 3rd, left eye, T+. cornea steamy; pupil large, oval, fixed; pain; iridectomy. December, 1910, left lens opaque.

Present condition (February, 1911): Both eyes, O.D. not cupped; tension, both eyes, N; some lens opacities; field, right eye, good; vision, with glasses, right eye, 6/9 with +2 and +2.5 170° and J<sub>1</sub> with +6; left eye, 6/60. No pain; no coloured rings.

Case 47.—65, 87, 230, Or., 1908.—D. M., male, aged 64. Admitted April 6th, May 6th, November 26th, discharged April 14th. History: In 1906 cataract extraction without iridectomy right eye; cataract, immature, left eye. In 1907, 6/6 with +8 and J<sub>1</sub> with +12. In March, 1908, right eye, 6/9 with +9; web; left eye, lens opacities. Treatment: April 7th, right eye, needling; April 10th, disc cupped, T+, field reduced greatly; left eye, field nearly full. May, iris torn away (iridectomy), escape of vitreous. November, V=less than 6/60 with +8 and J<sub>8</sub> with +16, field much reduced; left eye, lens opacities, V=less than 6/60. Iridectomy. prophylactic, left eye. Extraction, left eye, November, 1910.



Present condition (March 14th, 1911) (patient aged 67): Good coloboma, scar rather wide, not pigmented; left eye, good coloboma; tension, both eyes, N; right eye, O.D. wrinkled up; left eye, some irregular capsule material, O.D. appears healthy, vessels small; vision, with glasses, right eye, H.M. only; left eye, 6/9 (5). No pain.

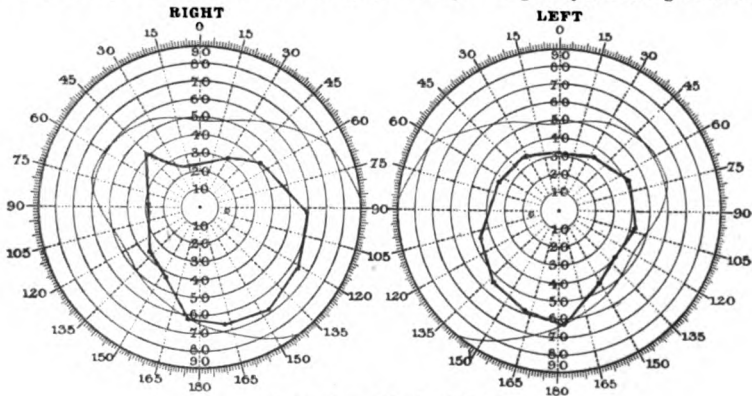
Case 48.—158, E., 1908.—E. P., female, aged 53. Admitted July 23rd, discharged August 20. History: Iridectomy, left eye, eight years ago; spots and floatings, right eye: worse during the past eight months. C.O.A.: Iris and pupil, right eye, small, pupil N; tension, +; O.D. slightly cupped; vision, 6/5 with -1.25 sph. and -1.0 cyl. 115°. Treatment: Iridectomy; hyphæma, after a blow; cleared up completely. C.O.D.: Ten-

sion, right eye, N; vision, 6/12 with  $+1$  sph. and  $-4.5$  cyl. and  $J_2$  with  $+1.5$  sph. and  $+4.5$  cyl., unchanged in 1909. Left eye, 1909, Tn, V= $6/60$ .

Present condition (March 14th, 1911): Both eyes, wide, clear coloboma; right eye, O.D. small, reddish, cupped in centre, with vessels definitely bent, T. slightly higher than in left eye; left eye, O.D. whiter, no cupping, some choroidal atrophy around; Tn (?full); right eye, striate opacity in coloboma, as shown in figure;

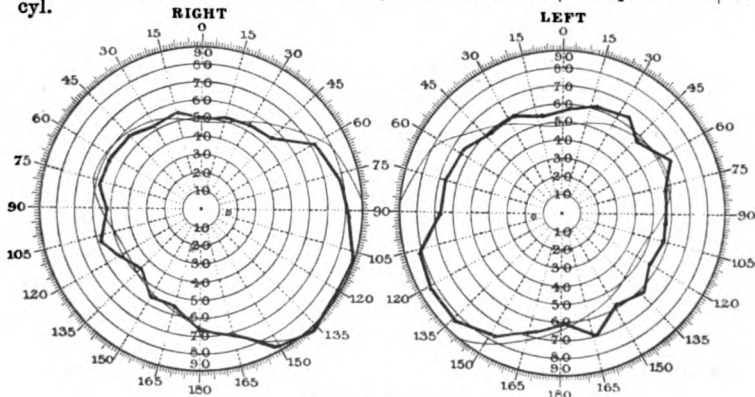


vision, without glasses, both eyes, 6/60; with glasses, right eye, 6/12 with  $-1$  sph. and  $-5$  cyl and  $J_4$  with  $+2.5$  and  $-5$ ; left eye, 6/12 (3) with  $-2$  sph. and  $-4$  cyl. and  $J_4$  with  $+7.5$  and  $-4$ . Right eye, occasional bulbar and supra-orbital pain; left eye, no pain. Right eye, coloured rings every fortnight or so; left eye, no rings. Right eye, floating bodies.



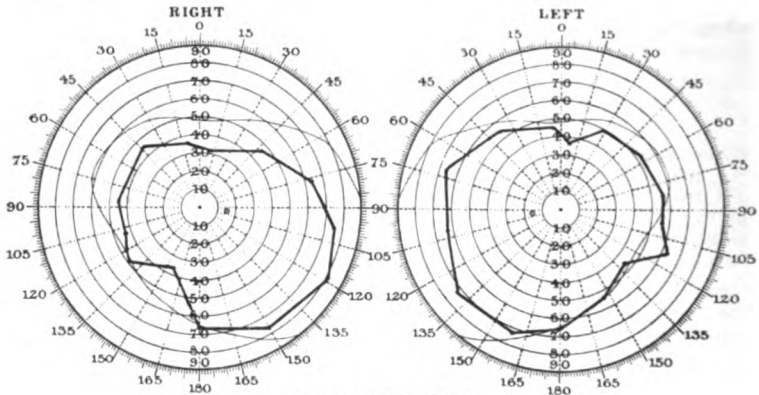
September 22nd, 1908.

1908. Iridectomy, July 25th; fields taken September 22nd. V=right eye, 6/18 with  $-4.5$  cyl. 100°. October 27th, Tn, disc not cupped. Right eye, 6/12 with  $+1$  sph. and  $-4.5$  cyl. and  $J_2$  with  $+1.5$  sph. and  $+4.5$  cyl.



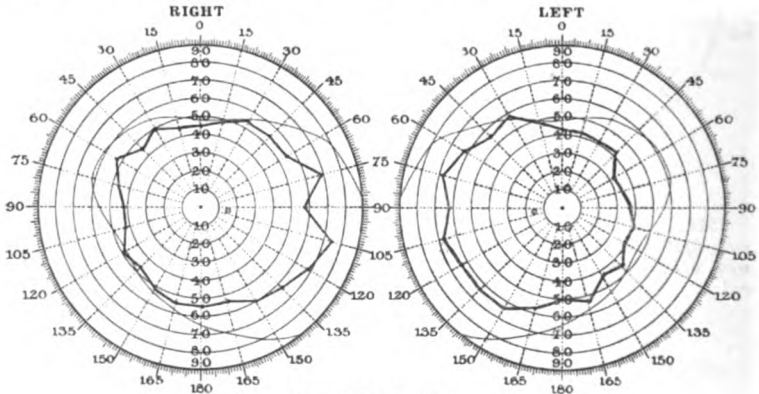
July 13th, 1909.

1909. February 2nd, right eye, 6/12 and  $J_2$ ; fields full. July 13th, right eye, 6/9 with  $-4.0$  cyl. and  $J_1$  with  $+1$  sph. and  $+4.5$  cyl. Left eye, 6/18 with  $-1.75$  sph. and  $-3.5$  cyl. and  $J_1$  with  $+3.5$  sph. and  $-3.5$  cyl.



January 11th, 1910.

1910. No record of vision; fields smaller.



March 14th, 1911.

1911. March 14th, right eye, 6/12 with  $-1$  sph. and  $-5$  cyl. and  $J_4$  with  $+2.5$  sph. and  $-5$  cyl. Left eye, 6/12 (3) with  $-2$  sph. and  $-4$  cyl. and  $J_4$  with  $+0.75$  sph. and  $-4$  cyl. Fields about the same as 1910.

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Case 49.—178, 1906.—A. S., female, aged 41. Admitted December 8th. History: Attacks of loss of vision, eighteen months, about once a month; subjective symptoms, coloured rings. C.O.A.: Cornea, both eyes, steamy; tension,  $+3$ , both eyes, both discs cupped, but specially the left, seen

with -30; fields fuller in right eye, nasal side of left lost, December 8th; vision, right eye, 6/12 on December 6th, left eye, 6/12. Treatment: December 14th, right eye, finger movements; left eye, iridectomy. Progress: February 1st, right eye,  $T+1$ ,  $V=6/24$ , field increased under eserine; left eye,  $T+$ ,  $V=6/36$ , great improvement since operation. February 20th, 1906, right eye, with -3 = 6/18; left eye, with -2 = 6/9 and  $J_1$  without lens.

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Case 50.—94, 1906.—G. H., male, aged 37. Admitted January 8th. History: Squint since birth, formerly fixed with right eye, now with left eye; movements good; very good till ten months ago, then right eye failed; at Christmas could only P.L.; duration of attack, ? one month; pain, after work (bootmaking); subjective symptoms, rings round light. C.O.A.: Cornea, both eyes, small, but fixes with left eye; opacity due to lime; iris and pupil, right eye, larger than left; tension, right eye, +2; left eye, +; right eye, disc definitely cupped; left eye, no cupping, field full; vision, right eye, P.L., left eye, 6/18 with +4, badly. Treatment: January 13th, right eye, upward iridectomy; January 17th,  $T+$ , pupil dilated. Progress: April 4th,  $V=6/18$  with right eye;  $V=P.L.$ , left eye. Advancement of right external rectus; little improvement.

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Case 51.—Or.—M. A. W., female, aged 69. History: Right eye blind two years; no pain; cornea trephined. April 18th, left eye, iridectomy.

Present condition (March 27th, 1911): Right eye, blind; left eye,  $T+$ ; vision, without glasses,  $T_n$ ; with glasses, 6/18 (some).

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Case 52.—89, 1906.—J. H., female, aged 68. Admitted February 23rd. History: Previous vision, sight, right eye, getting bad for past two years; duration of attack, two years; some pain, right eye. C.O.A.: Cornea, right eye, hazy; left eye, shallow a.c.; iris and pupil, right eye, semi-dilated, non-reacting, circumcorneal injection; left eye, reacts; tension, right eye, +2, disc red, not cupped; left eye, +1. Treatment: February 24th, right eye, upward iridectomy; February 27th,  $T$  decreased; left field full; right eye much contracted. Progress: March 8th,  $T+$ , both eyes; pupil, right eye, still very large, field still small; left eye, under eserine. C.O.D.: Vision, right eye, fingers at 6 ft.; left eye, 6/18 with +1.25 = 6/6, with +3 =  $J_1$ .

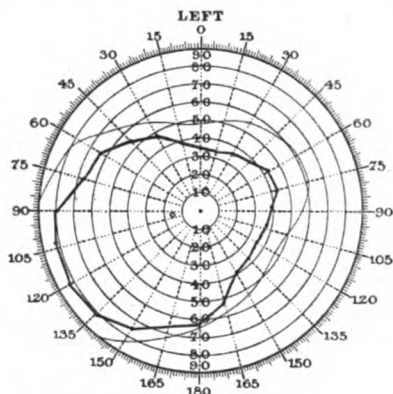
Present condition (March, 1911): Patient in Tooting Bec Infirmary, suffering from delusions; eyes no better.

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Case 53.—31, 1906.—W. C., male, aged 65. Admitted August 1st, discharged September 11th. History: July 26th, patient felt as if a piece of grit had got in right eye; came to hospital a week later; meanwhile, could only see a few feet with left eye; sight of right eye had been failing for three months before admission; very much inflamed and painful; coloured rings; floating bodies. Treatment: August 2nd, iridectomy,



right eye; August 8th, left eye, no pain. Progress: August 27th, Tn, cornea hazy; conjunctiva injected; V=fingers at 1 ft. September 10th, some vesicular keratitis. C.O.D.: Tension, right eye, N; vision, right eye, fingers at 2 ft.



Present condition (March 6th, 1911): Right eye, marked arcus senilis; dense white vascularised scar over pupil; good aperture at top; at night lights seen like brilliant stars; occasional pain in supra-orbital region; tension, +; vision, fingers, nasal only, at 18 ins. Left eye, pupil small and reacting; no pain; disc not cupped, lens opacities as indicated in



figure; vessels healthy; no retinitis; tension, N; field, see chart; vision, without glasses, 6/36; with glasses, 6/18 (with +2 sph.), with +2.5 sph. = 6/9, with +5.5 sph. = J<sub>2</sub>.

Case 54.—110, E., 1906.—L. H., female, aged 82. Admitted October 4th, discharged October 21st. History: Blindness in left eye, pain and headache, three years ago. C.O.A.: Right eye, shallow a.c., left eye, pain; iris and pupil, right eye, smaller than in left eye; left eye, fixed and dilated; tension, right eye, +; left eye, +3, green reflex; vision, right eye, 6/60, left eye, no P.L. Treatment: October 4th, right eye, iridectomy; left eye, enucleation.

Case 55.—1, H., 1906.—E. N., female, aged 58. Admitted January 1st and March 12th, discharged January 10th. History: 1897, iridectomy right eye, useless, ? P.L.; 1899, iridectomy left eye. Vision in left eye fair after operation; much increased during past eighteen months. Treatment: January 1st, left eye, iridectomy downwards; severe inflammation followed; much pain; no P.L. Progress: March, left eye, sclerectomy; pain relieved; general health better; sight uninfluenced: no headache to speak of.

Present condition (April 4th, 1911): Right eye excised in 1908 by Mr. Eason (blind and painful). Left eye, pupil dilated and fixed, attached iris; flitting bright sparks, clouds of light and darkness; pain in left eye until right eye was removed; this relieved it much.

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Case 56.—110, E., 1909.—F. H., male, aged 72. Admitted May 4th, discharged May 21st. History: Loss of sight both eyes; occasional pain in left eye, four years. C.O.A.: Left eye, anterior staphyloma; iris and pupil, right eye, pushed forward and displaced upwards, lens opacity; tension, both eyes, +; vision, right eye, no P.L. Treatment: May 9th, left eye, excision.

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Case 57.—65, 1907.—D. F., female, aged 44. Admitted August 16th, discharged August 28th. History: Blind in right eye for over two years; pain, last three weeks, considerable. C.O.A.: Cornea, right eye, some haziness and injection; iris and pupil, dilated and non-reacting; tension, +3; fundus not visible. Treatment: August 22nd, enucleation. Progress: August 25th, some effusion into cellular tissues.

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Case 58.—19, 1907.—H. B., male, aged 61. Admitted September 1st, discharged September 13th. History: Cataract, right eye, needed four years ago; glaucoma and blindness followed; cataract extraction, left eye, two and a half years ago; right eye occasionally painful. C.O.A.: Right eye, leucoma; left eye, clear; iris and pupil, right eye, blurred, no reaction, coloboma, irregular outline, fixed; left eye, iris deficient in upper half, fixed, no reaction; tension, right eye, +; right eye, fundus invisible; left eye, disc small (=tobacco amblyopia); vision, right eye, no P.L.; left eye, fingers at 2 ft.; with +13 = 6/24. Treatment: September 5th, right eye, excision; left eye, central colour vision good.

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Case 59.—14, 1909.—E. B., female, aged 58. Admitted September 6th. History: Operation, right eye, ? for glaucoma by Mr. Hardy six years ago; sight not improved; much increase of dimness last two months; painful during the past week. Left eye, vision worse; Tn, V=6/36 and J<sub>4</sub>; unable to read for many months. C.O.A.: A.c., right eye, hyphæma; left eye, N; cornea, right eye, steamy, oval, sensitive; left eye, some lens opacities, lens pigmented; iris and pupil, right eye, medium size, muddy; coloboma; left eye, posterior synechiæ; tension, right eye, +2; vision, right eye, faint P.L.; left eye, 6/36 with +2 cyl. = 6/18, badly, fundus not seen, disc not cupped. Treatment: Right eye, eserine, no operation. Progress: Improved. C.O.D.: 1910, February 1st, field reduced to one-half. Iridectomy. March 8th, field improved.

Present condition (March 14th, 1911): Iris and pupil, right eye, bulb shrunken and disorganised; left eye, no pain; tension, left eye, Tn; field, left eye, full; vision, right eye, no P.L. Excision refused by patient.

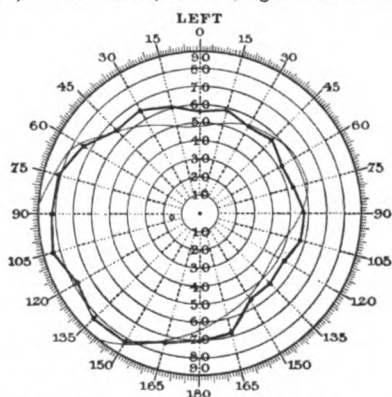
Case 60.—14, H., 1906.—W. B., male, aged 63. Admitted January 22nd. discharged February 4th. History: Left eye blinded by football twelve years ago, very painful at the time; duration of attack, five months; right eye painful; pain, three weeks ago, intense; subjective symptoms, vomiting, coloured rings. Treatment: January 22nd, right eye, iridectomy.

Present condition (April, 1911): By letter—Patient has been quite blind for more than four and a half years.

Case 61.—61, 1906.—T. D., male. Admitted May 6th. History: Iridectomy both eyes three years ago; vision good for eleven months after; admitted again one year later; for the last two years sight getting worse. C.O.A.: Both eyes, blepharitis and mucopurulent discharge; nebulæ; iris and pupil, right eye, irregular and almost occluded, lens ? opaque, iris drawn up and adherent; left eye, pupil large and dilated, aphakia; iris and capsule adherent; tension, both eyes, +: right eye, no reflex; vision, left eye, hand movements. Treatment: May 17th, iridectomy downwards, right eye. Progress: May 21st, hyphæma. C.O.D.: Vision, not improved; good aperture, downwards and inwards.

Case 62.—154 and 244, Or., 1908.—D. B., male, aged 45. Admitted August 10th, discharged August 20th. History: Iridectomy right eye two years ago; V=hand movements. Iridectomy left eye March, 1907; now unable to read even with magnifying glass; no pain; subjective symptoms, rainbows. C.O.A.: Left eye, disc cupped; vision, right eye, hand movements; left eye, fingers at 5 yds. Treatment: Left eye, sclerotomy. Progress: October 12th, Tn; cystoid scar; sight worse, 6/60; field much decreased. December 17th, acute pain; T+1; pupil fixed; improved under eserine, etc. January 12th, sclerotomy, left eye, C.O.D.: P.L.

Case 63.—89, E., 1909.—S. G., female, aged 67. Admitted August 30th,



discharged September 18th. History: May, acute bronchitis, sudden pain on rising from bed; duration of attack, for past four months rainbows

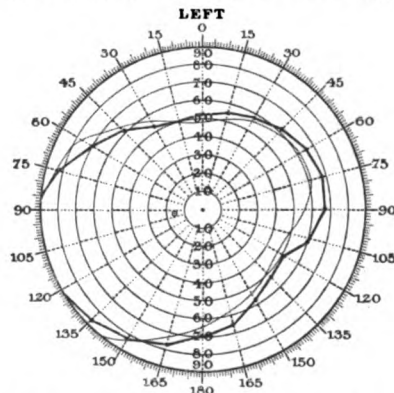
and dimness of vision; violent pain in left frontal region and behind ear; rainbows. C.O.A.: Cornea, left eye, steamy; iris and pupil, left eye, discoloured, dilated; tension, right eye, N; left eye,  $+2$ ; vision, left eye, P.L. and H.M. Treatment: August 31st, left eye, iridectomy. Progress: September 9th, left disc cupped; T $+$ ; no P.L. C.O.D.: Vision, left eye, P.L.

Present condition (March 28th, 1911): Iris and pupil, right eye, well under eserine; left eye, slightly divergent, some calcareous material at edge of the coloboma; tension, both eyes, N; vision, without glasses, right eye, 6/24; with glasses, 6/9 with  $+4$  and J<sub>1</sub> with  $+8$ ; left eye, P.L. Right eye, coloured lights and stars; no headache; no mistiness; floating bodies. Left eye, never painful. Field, see chart.

Case 64.—57, E., 1909.—W. D., male, aged 74. Admitted September 1st and December 13th, and discharged October 8th and January 4th, respectively. History: Sight of right eye gradually failing for nine months; occasional headache and pain during past week. C.O.A.: Cornea, right eye, steamy; iris and pupil, discoloured, normal size. sluggish; deep a.c.; tension,  $+$ ; vision, P.L. Treatment: September 1st, right eye, iridectomy; continued pain; T $+$ ; no P.L. October 4th, left disc cupped; field full. Progress: September 23rd, right eye, iridectomy. C.O.D.: December 13th, right eye, excision.

Present condition (March 24th, 1911): Right eye, excision. Left eye, vessels excentric and bent, but no obvious cupping; with glasses, J<sub>16</sub> with  $+5$  sph., 6/24 (2); not improved by glasses; clearer with  $+1$  sph.; constant pain in head, right side, since discharge; sight misty; no coloured rings or specks.

Case 65.—197, Or., 1908.—E. L., female, aged 49. Admitted October 14th, discharged October 31st. History: Vision failing for a long time;



on admission was "quite unable to see" with right eye; no pain; no coloured rings, etc. C.O.A.: Both pupils small and fixed; does not fix

with right eye; tension, right eye,  $+1$ ; left eye,  $+$ ; fields, right eye, contracted. O.D. a little cupped; left eye, full; vision, right eye, 6/18 (some) with  $+3$  sph. Treatment: October 16th, sclerotomy and iridectomy. Progress: Prolapse of iris. C.O.D.: 1910, Excision, because blind and painful.

Present condition (March 27th, 1911): Right eye excised. Left eye, tigroid fundus with retinal vessels over it; outline of O.D. blurred; veins congested; quite white; vessels rather bent at edge, but not discontinuous; no scleral ring; Tn:  $V=6/36$ ; with glasses, 6/24 with  $+2.0$  sph. and 0.75 cyl., and  $J_4$  with  $+4$  sph. Frontal headaches; nausea, but no vomiting; no coloured rings; no attacks of inflammation. Field, see chart.

Case 66.—122, Or., 1906.—E. C., female, aged 63. Admitted October 29th, discharged November 14th. History: Pain four to five years ago; sight dim and not much improved by glasses; coloured rings; sweating head; specks. C.O.A.: Tension, right eye,  $+1$  (with eserine), left eye,  $+2$ ; fields, both eyes, full, vision, both eyes, 6/9. Treatment: October 30th, left eye, iridectomy; prolapse of iris, with hæmorrhage. C.O.D.: No P.L.

Case 67.—53 and 122, Or., 1908.—L. S., female, aged 76. Admitted March 23rd and June 22nd, and discharged April 5th and July 25th, respectively. History: Duration of attack, five weeks; headache, no ocular pain; spots and coloured stars. C.O.A.: Both eyes, some anaesthesia; shallow a.c.; lens opacities, pupils react poorly to eserine; tension, right eye,  $+1$ ; left eye,  $+2$ ; both eyes, fields roughly normal. Treatment: In June, right eye, Tn;  $V=6/18$  with  $+2$  and  $J_1$  at 5 in. with  $+5$ . Left eye, iridectomy; in June  $V=6/36$  (1) with  $+1$  and  $J_1$  at 5 in. with  $+3$ . Both fields N. Right eye, iridectomy. C.O.D.: Right eye, 6/60 with  $+2$  and  $J_4$  with  $+5$ .

Present condition: Left eye, coloured rings and cloudiness. Right eye, vision very poor, described as "useless"; left eye, can read "close to": cannot see well at a distance, for example, across the street. No pain or inflammation.

Case 68.—45, 1906.—E. C., female, aged 19. History: Has worn glasses one year; sight failing, two years. C.O.A.: Cornea, both eyes, N; both discs cupped; myopic crescent, left eye; fields, right eye, much restricted; left eye, contracted; vision, right eye, with  $-1$  sph. and  $+2.5$  cyl.  $=6/12$ ; left eye, light at a few feet. Treatment: June 30th, right eye, iridectomy.

Present condition (March 3rd, 1911): Right eye, iris reacts, pupil dilated, shallow a.c.; tension,  $+$ , disc markedly cupped; vision, without glasses,  $V=6/36$ , fingers at 3 ft.; with glasses,  $J_4$  at 5 in.; attacks of pain every few weeks; never acute glaucoma; coloured rings. Left eye, tension,  $+3$ ; no P.L.; totally blind; no pain.

**Case 69.—24, H., 1906.—S. L.** Admitted February 19th, discharged March 29th. History: Three months' failure of vision; 1905, upward iridectomy; both eyes always misty, especially lately. C.O.A.: Right eye, lymph filling pupil; sees persons with difficulty; cannot read. Treatment: February 26th, right eye, iridectomy, down and out. C.O.D.: Tension, both eyes, —; vision, right eye, V=fingers at 2 in.; left eye, reads with +5.

**Case 70.—145, 1907.—A. P., female, aged 48.** Admitted November 21st, discharged December 3rd. History: Glasses since 16 years; left eye always weaker, losing sight for fifteen months; inflamed in October, 1907; patient very nervous. C.O.A.: Cornea, right eye, clear: left eye, steamy; iris and pupil, right eye, N: left eye, medium, irregular, fixed: muddy; fields, right eye, some contraction, except on nasal side; left eye, nasal and temporal narrowing; vision, left eye, fingers at 4 ft. Treatment: November 25th, left eye, iridectomy. C.O.D.: Tn: vision, fingers at 4 ft.

**Case 71.—169, 1907.—J. S., male, aged 62.** Admitted March 26th, discharged April 15th. History: Pain in left eye, worse during last month. C.O.A.: Cornea, clear; a.c. deeper in left than in right eye; iris and pupil, right eye, irregular, react; left eye, no reaction to light; lens opaque: tension, left eye, +2; vision, P.L. Treatment: March 28th, left eye, iridectomy. C.O.D.: Vision, right eye, with 1.5=6/9; left eye, hand movements; defective fixation.

**Case 72.—75, 1906.—B. F., female, aged 44.** Admitted July 6th. History: Sight of left eye failing, past eight months. C.O.A.: Shallow anterior chamber; iris and pupil, dilated, no reaction, discoloured: right eye, disc normal; left eye, T+, disc cupped and white: field, left eye, not ascertainable; vision, right eye, good P.L.: left eye, no P.L.. Treatment: July 9th, iridectomy, left eye. July 12th, pain relieved; T—.

**Case 73.—100, 1906.—S. J., male, aged 52.** Admitted August 10th, discharged August 28th. History: In 1902, pupils fixed and dilated, discs cupped, shallow a.c., T+1, fields limited peripherally; double iridectomy. 1903 and 1904, eserine. 1905, double iridectomy downwards. C.O.A.: Tension, both eyes, +1, variable; fields, peripheral vision only. Treatment: Eserine, 1 per cent. t.d.s. C.O.D.: Both eyes, T+-. Double optic atrophy.

Present condition (March 3rd, 1911): Right eye, iris and pupil, wide



coloboma (see figure); tension, Tn: field, disc cupped, fundus pigmented;

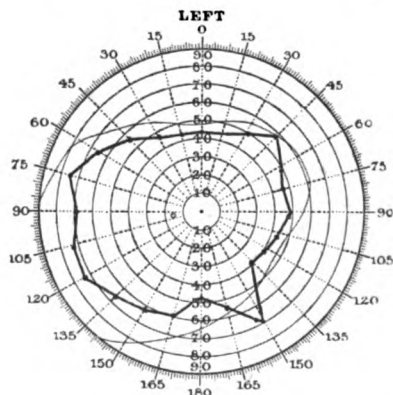
vision, P.L.; projection from above and rather to left and below; neuralgic pain. V=P.L. Left eye, wide coloboma (see figure); tension,



Tn; field, projection from above only, sector lens opacities; hand movements in line of vision only; no nasal or temporal field; neuralgic pain. V=H.M.

Case 74.—126, 1906.—M. N., female, aged 58. Admitted January 8th, discharged January 22nd. History: Acute glaucoma ten years ago; "saw white snake" four months ago; had pricking pain in left eye for two days. C.O.A.: A.c., both eyes, shallow; cornea, both eyes, dim at edges; right eye, old iridectomy, atrophy; left eye, large, fixed, irregular pupil; tension, both eyes, +; right eye, disc not seen, some nasal limitation of field; left eye, disc oval, and somewhat cupped, more nasal limitation; vision, right eye, 6/12; left eye, 6/24; good P.L. Treatment: January 10th, left eye, upward sclerotomy.

Case 75.—108, Or., 1906.—F. J. W., male, aged 60. Admitted October 1st, discharged October 24th. History: Failing sight, 4—5 years, especially in left eye. C.O.A.: Both pupils irregular and fixed by adhesions. Treatment: October 2nd, right eye, iridectomy; October 9th, left eye, iridectomy. Progress: Sent out wearing, right eye, plane, left eye, +12 for distance. Later, further operation, 1908, February, left eye, cataract extraction.



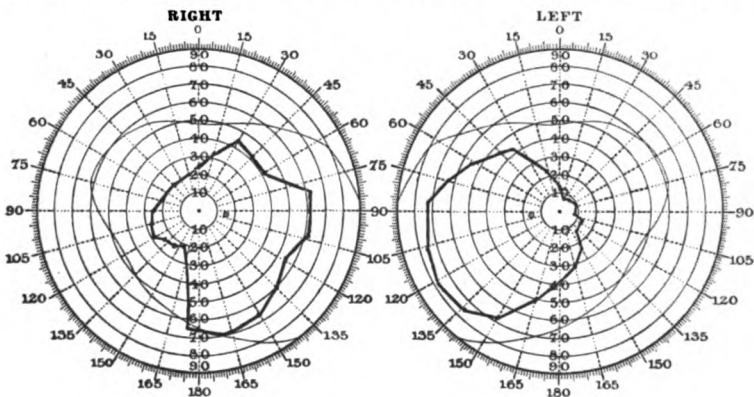
Present condition (March 28th, 1911): Right eye, shallow anterior chamber; good coloboma, with horizontal thread of fibrin across it; left eye, iris drawn up; small opening at upper part, all the rest occluded by web; tension, right eye, Tn, no reaction to light, O.D. not

seen; left eye, T+1; fields, right eye, central mature lens opacity; left, good fundus reflex; O.D. invisible; vision, right eye, perfect projection, 6/60, J<sub>8</sub> without glasses; with glasses, 5 in.; distant vision in daylight only; no pain or inflammation. Left eye, 6/24 (3) with +12 sph. and cyl., and J<sub>4</sub> with +14 and +1 at 5 in.; painful at night, sometimes; cannot read at all. Field, see chart.

Case 76.—87, 1908.—E. H., female, aged 65. Admitted February 29th, discharged March 17th. History: Duration of attack, four months; sickness, frontal headache, misty sight; sickness recently. C.O.A.: Right eye, shallow anterior chamber; left eye, normal anterior chamber; lens and O.D.; right eye, lens opacities, T+; left eye, T+; fields, right eye, much diminished; left eye, some nasal contraction; vision, right eye, 6/60, left eye, 6/9. Treatment: March 2nd, right eye, iridectomy. C.O.D.: Tension, right eye, N. March 27th, vision, fingers at 2 ft.; June, left eye, 6/9 with +1.5 sph.

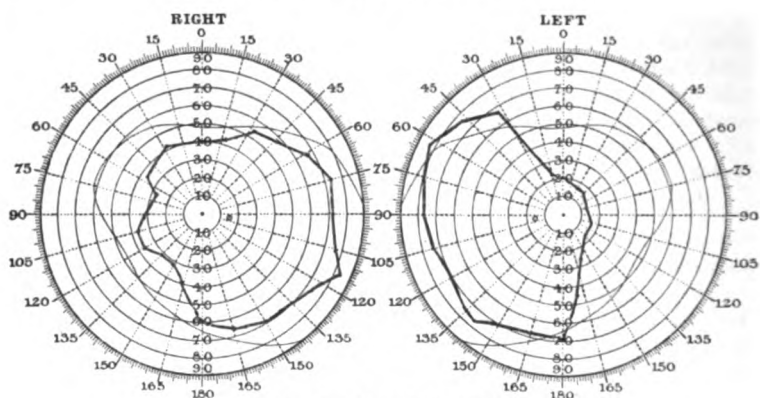
Present condition (March 24th, 1911): Vision, with glasses, right eye, 6/60. Left eye, tension, N; fields, full, O.D. cupped; vision, without glasses, 6/36; with glasses, 6/6 with +1.75 sph. and J<sub>2</sub> with +5.5 sph. Pain in both eyes ever since operation, especially in wet weather; giddiness; spiky moving figures round lights in left eye.

Case 77.—145, 1908.—E. N., female, aged 73. Admitted October 4th, discharged October 27th. History: Glasses for ten years; never any eye disease before; failing sight one year; no pain, photophobia, or lachrymation. C.O.A.: Both eyes, shallow a.c.; pupil, contracted by eserine; discs cupped; vision, right eye, 6/18; left eye, 6/24. Treatment: October 5th, left eye, iridectomy upward and outward. Progress: October 19th, right eye, iridectomy. October 26th, flashes of light and figures. C.O.D.: Tension, right eye, T+; left eye, Tn; vision, right eye, 6/18; left eye, 6/36.

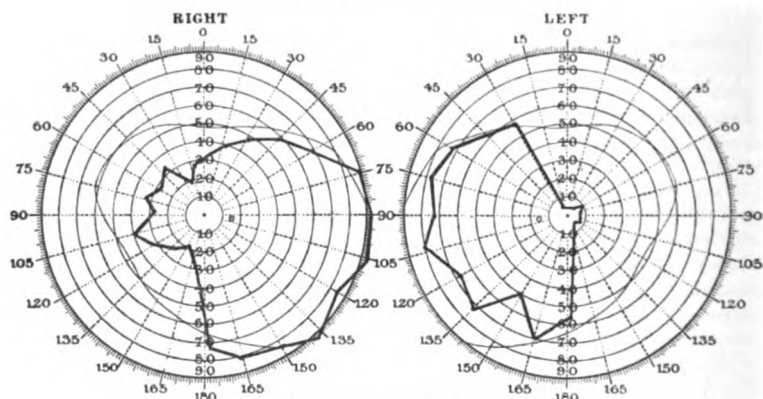


E. N., September 25th, 1908.

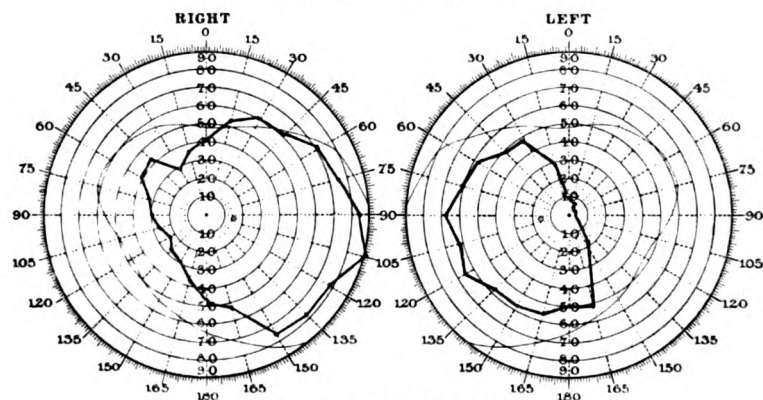




E.N., October 26th, 1908.



E. N., February 19th, 1909.



E. N., March 17th, 1911.

**Present condition:** Right eye, coloboma upward; scar 1 mm. behind c.-s. junction; left eye, coloboma upward and outward; scar at limbus, pigmented at outer end; tension, both eyes, N; fields, right eye, slight nasal contraction; left eye, small no nasal field at all; vision, without glasses, right eye, 6/36; with glasses, with +4.5 sph. and +1.5 cyl. = J<sub>4</sub>. Left eye, without glasses, fingers at 18 in.; with glasses, 6/36 with +1 sph. O.D. oval, shrunken, pale, slightly cupped above; lens opacities (radial striæ) both eyes; no fundus degeneration.

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**Case 78.**—161, Or., 1908.—M. B., male, aged 55. Admitted August 27th, discharged September 12th. History: Glasses one year; sight failed rather suddenly six months ago; has been treated at Royal Eye Hospital; no pain. C.O.A.: Iris and pupil both eyes, small, round, reacting; tension, +; fields, much contracted; vision, 6/60. Treatment: August 28th, right eye, iridectomy. C.O.D.: Tension, right eye, N; left eye, +1; vision, both eyes, 6/60.

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**Case 79.**—56, Or., 1909.—J. E., male, aged 51. Admitted March 29th, discharged April 15th. History: Duration of attack, two years; mistiness; coloured rings. Treatment: March 30th, left eye, iridectomy. C.O.D.: Vision, right eye, 6/9, H.M., +0.5 sph. and J<sub>4</sub> with +2.5 and +2.0; left eye, 6/60, fingers at 1 yd., and 6/24 with +2.0 sph. and -5.5 cyl.

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#### NOTE.

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The above paper was written in the spring of 1911. On visiting Southampton Free Eye Hospital later in the year, I found that Mr. Zorab had not only already suggested the operation of lymphangioplasty for chronic glaucoma, but had actually performed it.

He has published an illustrated account of his method, together with the preliminary reports of seven cases, in *The Ophthalmoscope*, May, 1912 ("The Reduction of Tension in Chronic Glaucoma"). As the author says, "Considering the very short time that has elapsed since the performance of the first operation, it would be premature to accept the results as permanent"; but it seems to be clear that if the silk is properly inserted, it will remain in situ without causing irritation.

No details are given as to the effect of the operation upon the patient's vision, but tension seems to be satisfactorily reduced, and the technique is not difficult. It is to be hoped that other surgeons will give this method a trial.



# THE ÆTIOLOGY OF MELÆNA NEO- NATORUM, WITH SPECIAL REFERENCE TO DUODENAL ULCER.

BEING A THESIS FOR THE DOCTORATE IN MEDICINE  
OF CAMBRIDGE UNIVERSITY.

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By


WILSON TYSON, M.A., M.D. Cantab.,  
Surgeon, Lowestoft Hospital.

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## INTRODUCTION.

THE condition known as *Melæna Neonatorum* is one of considerable rarity, occurring, according to some authorities, about once in a thousand births, and the prognosis is extremely grave. Although its occurrence is fully recognised, the ætiology and pathology are still very obscure, and many theories have been advanced to explain the condition. In the opinion of the writer, it has not yet been sufficiently appreciated that the condition is a manifestation of a micro-organismal disease, and that it is frequently associated with an acute ulcer of the duodenum (or of the stomach).

Having recently had under my own care two severe and fatal cases of this disease, I have, in addition to investigating the literature of the subject, analysed all those cases which have occurred at the Queen Charlotte Hospital during the years 1896



—1910, and the results have been incorporated in this paper; the general question of the ætiology of the condition has also been considered, apart from the cases due to duodenal or gastric ulcer.

The Reports of the Queen Charlotte Hospital for the fifteen years referred to contain records of 21,119 births; in 1,358 of these cases (see appendix) the infants were either still-born or died in the hospital, and in only 17 of these was the occurrence of melæna noted. These figures would tend to confirm the statement contained in Moynihan's work on Duodenal Ulcer that melæna neonatorum occurs about once in a thousand live births.

I wish to take this opportunity of acknowledging my indebtedness to the Medical Committee of the Queen Charlotte Hospital for permission to make use of the medical reports of that institution, and also of expressing my sincere thanks to them for their further kindness in allowing me to publish the notes of the cases thus obtained. My best thanks are also due to Dr. Herbert French for kindly furnishing me with the notes of Case 21, which was recently admitted into Guy's Hospital.

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#### HISTORICAL OUTLINE.

The history of melæna neonatorum may be considered in connection with the whole syndrome of hæmorrhage in the new-born. The first observation on a case of this nature appears to have been that of Mauriceau<sup>56</sup> in 1682. This was a case of umbilical hæmorrhage. Since that date the developments in connection with this subject may be divided into four groups (51a):

1. Period of Confusion.
2. Clinical Period.
3. Anatomical and Pathological Period.
4. Laboratory Period.

The last of these may be divided into two stages, bacteriological and humoral.

*First Period, 1723—1825:*

This extends from Ebarth to Hess. In 1723 Ebarth<sup>21</sup> recorded the first detailed observation. It appears to have been a case of spurious melæna.

Watts, in 1752, and Underwood,<sup>89</sup> in 1786, recorded cases of umbilical hæmorrhage.

Brebisius<sup>10</sup> recorded four cases, two of which were cases of intestinal hæmorrhage. He sought an explanation of the condition in the asphyxia due to unnaturally prolonged labour.

Vogel,<sup>90</sup> in 1795, also admitted the influence of pressure during labour leading to venous stasis. He thought the compression of the umbilical vessels resulted in the turgescence of the mesenteric vessels, and consequent melæna.

La Fourie,<sup>48</sup> of Montpellier, recorded a case of hæmatemesis in a newly-born infant, and considered that the condition was allied to hæmophilia.

In the Archives de médecine, chirurgie et pharmacie, de la Société de Médecine suisse, 1816, vol. 1, page 57, a writer states that hæmorrhage of the newly born is the result of a plethora.

This theory was later held by Landau<sup>49</sup> and Kling,<sup>45</sup> but combated by Schmidt.

Cheyne<sup>14</sup> published an observation on a case of umbilical hæmorrhage with an anatomical description of the parts as seen in a post-mortem observation.

Prout<sup>65</sup> published a very interesting observation on three infants of one mother, all of whom succumbed to umbilical hæmorrhage.

*Second Period, 1825—1838:*

The second, which might also be termed the period of congestion, commenced with the monograph of Hesse,<sup>35</sup> in 1825. He recalls the observation of Brebisius, and suggests the hindrance to respiration at birth as an explanation. He mentions also the observations of Storck and Vogel (*vide supra*), and explains their cases as being due either to stasis on account of the obliteration of the umbilical veins, or to passive congestion due to obliteration of the arteries. He attributes the melæna

either to rupture of vessels in the mouth, nose, pharynx, lungs, as was thought by Brebisius, Storck, and Vogel, or ascribes the origin of the blood to the uterus, as was thought by Ebarth.

Oslander, in 1819, had shown that the foetus can make efforts at deglutition within the uterine cavity.

Billard,<sup>8</sup> in 1825, in a monograph on the mucous membrane of the digestive tract mentions ulcerations of the gastrointestinal tract as a cause of melæna. He notes a condition of folliculitis leading to ulceration, and an active or passive congestion leading to effusions of blood in the submucous layer. To explain the umbilical hæmorrhage he suggests an increased permeability of the vessels of the umbilicus, and cites statistics to prove it.

Orfila<sup>64</sup> deals vaguely with the question from the medico-legal point of view, and mentions cases where intestinal ulcerations were the cause of the melæna.

Rahn Escher,<sup>67</sup> in a clinical study published in the *Gazette médicale*, 1835, believed the condition to be due to a debilitated state of the parents.

*Third Period, 1838—1874:*

In 1838 Gendrin<sup>29</sup> dealt with the condition from an anatomico-pathological point of view.

Siebold<sup>77</sup> attributes melæna neonatorum to erosions and ulcerations of the intestine brought about by the amniotic fluid swallowed by the infant.

Barrier<sup>6</sup> gave a résumé of the question, and agreed with the views of Billard (*vide supra*) as to primary congestion, attributing the condition to primary ulceration. The work of Rilliet on the subject is of great importance, and nearly all subsequent authors have been influenced by his views.

Von Busch<sup>11</sup> described a case of melæna neonatorum due to a perforating ulcer in the greater curvature of the stomach (verified by post-mortem examination).

Hecker and Buhl<sup>33</sup> state that intestinal ulceration was the sole cause of melæna neonatorum, but they remain silent as to the pathology of the ulceration. Spiegelburg and Bohn<sup>81</sup> re-

ported further cases of melæna neonatorum due to ulceration—a folliculitis due to a closure of the gland ducts and secondary ulceration. Steiner<sup>83</sup> held that the ulceration was produced by fatty degeneration of the vessel walls. Rehn<sup>89</sup> considered the ulcerations were produced by a necrosis of the mucous membrane of the stomach caused by the gastric juice.

Landau<sup>50</sup> established the almost constant co-existence of melæna neonatorum with ulceration. His view was that there occurred a thrombosis of the umbilical vein or artery. From this thrombus emboli were detached, and these were the actual cause of the ulceration and consequent hæmorrhage.

Kling<sup>46</sup> agreed that this theory would account for some of the cases, but not for all. Recently several other cases have been observed where the melæna neonatorum was due to intestinal ulceration.

Dietel,<sup>19</sup> in 1896, and Latarjet,<sup>47</sup> in 1904, record such cases.

In this country, Radford,<sup>86</sup> Ray,<sup>88</sup> Manley,<sup>84</sup> Simpson,<sup>78</sup> and Hardy,<sup>32</sup> all recorded cases of melæna neonatorum.

Habershon,<sup>31</sup> Wilson Fox,<sup>93</sup> and Croom,<sup>17</sup> recorded cases, but without drawing any conclusions as regards their ætiology or pathology.

In America, Bowditch,<sup>9</sup> Minot,<sup>59</sup> Smith,<sup>79</sup> and Jenkins,<sup>41</sup> dealt with the subject. The last of these collected 178 cases.

#### *Fourth Period:*

In this period the influence of Pasteur and his school led observers to attribute the condition to microbic origin. Bartels<sup>7</sup> was the first observer to give as a cause of umbilical hæmorrhage an infection of the blood.

Scanzoni<sup>74</sup> gave an interesting account of the relationship between these hæmorrhages and puerperal infection.

Klebs, in 1874, described an organism that he found not only in the blood, but also in the tissues in such cases, and he named the organism *Monas hæmorrhagica*.

Eppinger,<sup>22</sup> who held the same views as Klebs, described in 1878 a fatal case of hæmorrhage in an infant in which he found the *Monas hæmorrhagica* in the tissues.



Henoch<sup>34</sup> goes further and attempts to explain how the organisms produce hæmorrhage. He believes that the masses of micrococci can act as emboli in the veins.

Rehn<sup>70</sup> discovered in the blood of newly-born infants, who had succumbed to hæmatemesis and melæna, a microbe which resembled the *Bacillus pyocyaneus*.

Schaffer<sup>75</sup> recorded a case in which the *Bacillus pyocyaneus* and *Staphylococcus pyogenes aureus* were associated, and in another observation he described a duodenal ulceration which he attributed to the *Bacillus lactis aerogenes* present in the spleen.

Gärtner,<sup>27</sup> in 1893, described a specific organism now known by his name which he found in cases of melæna neonatorum, and which on injection into animals produced melæna. The bacillus described by Gärtner is short, grows well on ordinary media at 37° C., producing a large amount of gas. It stains readily by the aniline dyes. It differs from the *Bacillus typhosus*, *Bacillus coli*, and *Bacillus aerogenes*. Although Gärtner believed this was the specific organism of melæna neonatorum, not all observers were in agreement with him.

Neumann<sup>63</sup> and Luyt<sup>53</sup> believed the causative organism to be the *Streptococcus pyogenes* which they found in the organs of their cases.

Spiegelberg<sup>80</sup> mentions two cases where the hæmorrhage followed abscess in the new-born.

Comby<sup>15</sup> described an epidemic of vulvo-vaginitis in infants accompanied in certain cases by grave hæmorrhages.

By the side of these microbial theories the views that the condition is due to a syphilitic infection must be considered. Several observers have ascribed the condition to syphilis. Among them may be mentioned Grynfeldt,<sup>30</sup> Hill Shaw,<sup>36</sup> and Conroy.<sup>16</sup> Other observers, who after careful examination have not been able to find any other cause of the condition, have attributed it to hæmophilia.

Milton Lewis<sup>58</sup> made a résumé of the subject based on 185 cases of melæna neonatorum found in the literature, and came to the conclusion that the condition was produced by a

lesion of the brain during birth. Pomorski, Kundrat, and Von Preuschen hold the same view.

Demelin,<sup>18</sup> in his article on hæmorrhages in the new-born, sums up the question very well. He says the condition may be due to one of three groups of causes—

1. Accidents during birth.
2. Disturbances in the circulation during the establishment of pulmonary breathing.
3. External conditions acting on the infant as on the adult.

In this group the principal cause is infection.

With regard to microbic infection, this author states that it may be due to various organisms, principally the *Streptococcus* and *Staphylococcus pyogenes*.

One of the most recent works on the whole subject is that of D. W. L. Stowel.<sup>85</sup> He holds that hæmorrhage in the new-born constitutes only a symptom. He has carefully studied the various causes of the condition, and reports a series of personal observations.

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#### ÆTIOLOGY AND PATHOLOGY.

A number of different theories have been advanced to explain the causation of this condition. These will now be considered.

I. *Cerebral Injury*.—With a view to disposing of the theory of Von Preuschen and Pomorski that many, if not all, cases of spontaneous hæmorrhage are secondary to cerebral injury, notes of all the cases where cerebral or meningeal hæmorrhage occurred at the Queen Charlotte Hospital have been made. Twenty-four cases, in twenty-three of which a post-mortem was made, occurred, and in no case was the cerebral hæmorrhage associated with gastro-intestinal hæmorrhage. In all the cases but three the labour was abnormal; in one of these there was a marked dent over the right parietal bone, and the child died of fits,

whilst in another there was also congenital morbus cordis and atelectasis, but no abdominal hæmorrhage, and in the third there was also atelectasis. No cerebral lesion was present in any of the fatal cases of melæna.

II. *Backward Pressure.*—The idea that melæna is due to backward pressure from heart lesions or atelectasis (Cautley,<sup>13</sup> page 128) is not supported by an examination of the cases in which either of these conditions was present. In the different cases a variety of heart lesion was present, but no case of melæna occurred. In one case both a congenital heart lesion and a cerebral hæmorrhage were present, but no melæna occurred.

III. *Landau's Theory.*—Landau<sup>51</sup> propounded the theory that melæna was to be found in weakly and premature infants where respiration was started with difficulty and delay. This condition favours stagnation and clotting of the blood in the umbilical vein. A clot is then detached either there or in the ductus arteriosus, and finally lodges in one of the arteries supplying the stomach or duodenum. In no case in the Queen Charlotte Hospital Records where atelectasis was described as being present post-mortem did melæna occur, and no condition of thrombus of the umbilical vein was present in the cases where this condition was specially looked for. The infants who suffered from melæna were full term children of quite average development, and, therefore, not of the type suggested by Landau as being likely to have any respiratory difficulty.

IV. *Relation to Syphilis.*—The question whether syphilis alone is a cause of hæmorrhage in these cases, or whether there is a complicating general infection, has been much discussed. Finkelstein,<sup>24</sup> from a study of seven cases, believes that the hæmorrhages are always due to a secondary bacterial infection. He says a true syphilis hæmorrhagica of the newborn does not exist.

Schwarz and Ottenburg relate a case where melæna occurred in a baby, where the Wassermann reaction was obtained, and where the treponema were obtained from the liver, where also a blood culture was sterile; yet they admit that a local infection

in the nose may have existed, and that toxic absorption from such a local lesion may be the ultimate cause of the loss of coagulability, which was present also in this case.

Townshend<sup>87</sup> says syphilis is undoubtedly a cause in some instances, this being so in three of his cases.

Holt<sup>88</sup> says syphilis is associated in a small proportion of cases, 2—6 per cent.

Mracek<sup>62</sup> found 14 per cent. of 132 cases of congenital syphilis suffering from hæmorrhage. Mracek believes in a special fragility of the blood vessels due to syphilis, etc. Evidence of vasomotor disease has not usually been found; probably these conditions simply prepare a suitable soil for the growth of organisms.

Still<sup>84</sup> says an association with syphilis does exist: endarteritis, probably syphilitic in origin, has been demonstrated in the small vessels in some fatal cases of melæna neonatorum. The frequency of this association, he says, has been exaggerated, and is actually very rare.

Cautley<sup>13</sup> (page 128) says congenital syphilis is regarded as an important predisposing factor.

Abt,<sup>1</sup> of Chicago (1903), found it present in two out of twelve cases, but Marchell,<sup>55</sup> of Toronto, obtained no evidence of it in either child or parent in 14 cases. It would appear, therefore, that syphilis is a predisposing cause and produces changes in the vessel walls favouring the growth of organisms and the onset of hæmorrhage.

In Case 10 of the series, where fatal hæmorrhage from the cord resulted, the condition of the liver, spleen, and retro-peritoneal tissue suggests syphilis as a contributing cause, whilst the cord was noted as soft and rather pus-like, denoting presence of sepsis. In Case 11, when the melæna was fatal, the placenta is described as syphilitic, the association of subperitoneal hæmorrhage beneath the umbilicus is here, too, suggestive of the existence of a septic condition of things.

V. *Trauma*.—In 19 out of 20 cases of melæna the labour was normal, the only exception being a case of delivery by forceps;

therefore, traumatism cannot be accounted a frequent cause. In only 6 out of 50 cases quoted by Townshend was the labour difficult. In contradistinction to this are the cases where massive hæmorrhages occur into the solid abdominal viscera. In the reports of such cases occurring at the Queen Charlotte Hospital there was in nearly every instance severe manipulation necessary to effect delivery. Here traumatism would seem to be an important factor, as also in the cases of intracranial hæmorrhage previously referred to.

VI. *Hæmophilia*.—The sexes were affected in the proportion of 9 males to 11 females, which is strongly against the suggestion that the condition should be one of hæmophilia, where the proportion is 13 males to 1 female. Other reasons against the hæmophilic origin are advanced in the section relating to Diagnosis.

VII. *Purpura*.—The occurrence in some cases of an accompanying purpuric rash may be regarded as evidence of the existence of a septic process, and may be compared to the purpuric rashes found in severe types of scarlet fever, measles, etc.

VIII. *Defective Blood Coagulation*.—Apart from the action of toxins or hyaline thrombi on the vessel walls, it has been shown by Schwarz<sup>76</sup> and Ottenburg that an altered blood condition due to defective coagulation is a pronounced feature in the causation of melæna in the newborn. The following notes of one of their recorded cases show that the blood failed to clot at all, and that transfusion of blood from the child's father resulted in an immediate alteration in the coagulation time to  $3\frac{1}{2}$  minutes with cessation of hæmorrhage; the bleeding subsequently recurred with a corresponding lengthening of the coagulation period to three hours:—

*Notes of Case*.—Male, full term; 4th child. Breast fed. Normal labour. No other bleeders. Abscess in breast. On 7th day black vomit after feeding. 8th and 9th day vomiting continued; stools loose and bloody. 10th day temperature 99°; ecchymosis over left spine. 11th day 20 c.c. horse serum injected; no effect. Transfusion from father. Hæmoglobin 30 per

cent.; after 12 minutes rose to 90 per cent. No clotting before transfusion. After transfusion, clotting in  $3\frac{1}{2}$  minutes, and bleeding ceased. Hæmorrhages returned, and child died eight days after transfusion; the coagulation time gradually lengthened, and was three hours on the third day before death.

Schwarz and Ottenburg conclude that: (1) Impaired blood coagulation is the immediate cause of uncontrollable hæmorrhage in the newborn; (2) Probably due to destruction of, or interference with, the production of thrombokinase; (3) Bacterial infection is the most frequent underlying cause.

*IX. The Toxæmias of Pregnancy, and other antenatal causes.*—Two of the cases reported by Moynihan were recorded by Waldeyer, who says the primary stages of the process leading to ulceration had commenced during foetal life. The occurrence of this antenatal pathological process would appear to be borne out by Case 11, where death occurred within half an hour of birth after a normal labour, and where the intestine was found full of blood. The placenta in this case was described as probably syphilitic. Toxins probably reached the foetus in utero via the placenta. The question of the existence of an antenatal or intranatal pathological condition to account for the occurrence of melæna neonatorum is a very obscure one. Professor Ballantyne in a letter to me on the subject stated that he was inclined to think that in some cases an antenatal, and in others an intranatal, *i.e.*, mechanical cause, existed, but as to its nature, he was not able to express any opinion.

No mention in the Queen Charlotte Hospital Records is made of the existence of any toxæmic condition in the mothers previous to delivery of those infants who suffered from melæna. In connection with this possible maternal fons et origo mali, it may be mentioned that in three of the series, sepsis after delivery did occur, and in one of these cases an inflamed breast was providing a supply of germ-containing milk for the suckling infant.

*X. Frequent Association with sepsis and presence of Acute Ulcer of Duodenum (or of Stomach).*—*Association of Hæmorrhage and Sepsis.*—First of all, in general pathology, the association of sepsis and hæmorrhage is fully recognised.

In 1897 Klein<sup>44</sup> examined the fluid taken from vesicles on the skin of a man who a few days previously had been skinning sheep which had died of puerperal septic œdema. He grew a streptococcus which, after injection into guinea-pigs, rabbits, and sheep, caused subcutaneous and intermuscular connective tissue hæmorrhages round the site of inoculation, along with blood effusions into the pleural and peritoneal cavities. "Septicæmia hæmorrhagica" is a well-known term in veterinary pathology for an important class of epizootic fevers, such as "barbone bufali," or buffalo disease. In India and Egypt hæmorrhages occur in horses and cattle in cases of strangles. Some cases of purpura are traceable to streptococcal infection. The acute hæmorrhages which accompany cases of faucial diphtheria are very fatal, streptococci and staphylococci being found besides diphtheria bacilli. Gastric and intestinal hæmorrhages occur after abdominal sections, and are probably septic in origin; erosions or small ulcers may develop; in some cases the hæmorrhage may occur from the mucous surface without any naked-eye breach of surface, or a raw surface may be present without any special blood vessel being opened. Probably a profound alteration in the blood, due in most cases to toxins of septic origin, is the chief factor in the production of these hæmorrhages. In splenic anæmia it seems to be proved that a toxin elaborated in the spleen causes hæmatemesis as a frequent symptom.

*Pathology of Hæmorrhage due to Sepsis.*—It may be well at this stage to make a few general remarks on the pathological processes resulting in hæmorrhages due to some infective process. It is necessary to recognise two orders of escape of blood:


1. By rhexis, by gross breach of continuity of the vessel wall.
2. By diapedesis, occurring in capillary vessels, without gross breach of continuity. Under similar circumstances and in the same area both varieties may occur. In many of the acute infections, hæmorrhages are found to be associated with the presence of hyaline thrombi in the capillaries which have an obstructive effect, arresting the blood stream, leading to local stasis at either side of the block, and producing minute hæmor-

rhagic infarcts. Adami<sup>2</sup> explains in this way the ulcer of the duodenum in cases of burns.

A preliminary hæmolysis or disintegration of corpuscles due to action of bacterial toxins precedes the formation of hyaline thrombi in these infectious disorders. In many infections we have to deal simply with direct toxic injury to the capillary endothelium. We may have (a) the specific action of toxic substances upon the capillary endothelium, with giving way of the same; (b) active growth of bacteria or infectious agents, within the endothelial cells, and lumen of capillaries, destruction of cells, and escape of blood. Both (a) and (b) may occur, the latter more particularly, in cases of bacteriæmia, in streptococcal and other terminal infections. Many of the hæmorrhages of infectious diseases accompanied by hyaline capillary thrombi may be of the nature of (a), *i.e.*, the localised coagulation of blood is secondary to the epithelial degeneration.

*Ulceration of and Hæmorrhage from the Duodenum is the result of a Toxic Condition.*—In burns an ulcer only occurs in the duodenum where septic processes in the burnt skin have developed; septic emboli might be conveyed from the infected area and produce hæmorrhagic infiltration which, immediately beyond the pylorus, would readily be converted into an ulcer by the action of the acid chyme. It is a *toxic ulcer*, and, therefore, analagous to the ulcer which occurs in septicæmia, uræmia, typhoid, erysipelas, and pemphigus.

Hunter<sup>40</sup> injected tolulyendiamine subcutaneously into dogs which were killed after 3—7 days, when the following changes were found: Even before opening, the duodenum was obviously inflamed, its walls turgid, swollen, and feeling doughy to the touch. On opening, inflammatory congestion of the mucous and submucous coats was seen, and the lumen of the canal was filled with a large quantity of clear inflammatory material. In other cases ulcers were found, the changes being most marked in the neighbourhood of the bile papilla; occasionally other parts of the small intestine were affected, especially the terminal portion of the ileum. Even after ligature of the common bile duct, ulcera-





tion of the duodenum was produced by the same means. Extensive scalds will produce hæmolysis similar to that produced by tolulyendiamine.

Lesser,<sup>52</sup> quoted by Hunter, describes marked changes in the blood after burns.

Gandy<sup>26</sup> has shown that congestion and hæmorrhagic erosion, which are the common precursors of ulceration in mucous membrane due to toxæmia, occur elsewhere in the alimentary canal, but are more readily converted into an ulcer in the case of the first part of the duodenum, probably because of the forcible ejection of the acid chyme through the pylorus.

Barie<sup>5</sup> considers that in uræmia, as a result of renal insufficiency, there are soon developed other supplementary paths for the elimination of the urinary poisons; these are established chiefly through the respiratory passages, by the skin, and perhaps by some of the serous membranes, but it is chiefly the alimentary canal which becomes the principal medium of excretion.

Stassano,<sup>82</sup> in his experimental work, supports this view of the excretory activity of the intestinal tract in cases of uræmia, and the special efficiency of the duodenum in the excretion of urinary toxins.

*Melæna Neonatorum is due to Sepsis.*—With regard to sepsis and the occurrence of melæna neonatorum, it is increasingly probable that the hæmorrhage is, in these cases, a manifestation of a micro-organismal disease. Holt<sup>37</sup> suggests that the organisms may act by producing a substance which destroys the epithelium of the small blood vessels akin to "hæmorrhagin" found by Flexner and Noguchi in rattlesnake venom.

Klebs,<sup>43</sup> in 1875, found a micrococcus in nine newborn children who had died of melæna, and cultures of it produced fatal hæmorrhagic lesions in young rabbits.

Bar<sup>4</sup> found streptococci in three cases.

In 1894 Gärtner published two cases of fatal melæna in which he found a short bacillus. Cultures were injected into the peritoneal cavity of puppies, producing a fatal gastro-intestinal hæmorrhage. A variety of other organisms (as previously men-

tioned in the historical section) have been discovered in this condition, *e.g.*, streptococci, staphylococci of various kinds, bacterium lactis aerogenes, diplococcus pneumoniae, Friedlander's pneumobacillus. In both of my own cases patches of necrosis of liver cells were present, markedly so in Case 2, indicating a toxæmic condition; in Case 1 there was also present a duodenal ulcer close to the pylorus, and this, in my opinion, may further be regarded as evidence of a toxæmic condition. In Case 2 organisms were present in the blood withdrawn from the vena cava, and pericardial fluid, and there were also present many gram-positive cocci in the tissues of the cord stump.

Blood cultures were also made in Cases 18 and 19, a negative result being obtained in the former, and in the latter the presence of bacillus coli was detected. As an additional reason for regarding melæna neonatorum as due to bacterial origin, we may instance the very definite course either to recovery or death within a limited time. This self-limited nature is characteristic of infectious diseases and suggests a relationship to them.

A further point suggesting an infective origin is the elevation of temperature observed in many cases. In 14 cases reported by Townshend<sup>86</sup> the temperature of these infants was studied, and in all but two was found to be elevated, and later, on cessation of hæmorrhage, to sink to normal, often being subnormal. The two supposed exceptions were not really such, as the temperature was not taken until the second stage of depression was reached. In 5 out of the 20 cases which I have collected pyrexia was present. The frequency of the affection in hospital in-patients as compared with its frequency among out-patients is suggestive of the infectious nature. In-patients, 45 out of 6,700 deliveries, or .67 per cent. Out-patients, 4 out of 4,000 deliveries, or .1 per cent. 29 cases out of 61 observed by Epstein, and 21 cases out of 190 observed by Rutter, were associated with sepsis (Holt,<sup>38</sup> page 102).

Rolleston shows that in many acute infections the micro-organisms or toxins produce acute changes, such as cloudy swelling, necrosis, leucocytic infiltration in adrenals previously

healthy; in some instances the damage is so acute that hæmorrhage occurs into the glands. It may be found at autopsy in a case of melæna, and may then be a further manifestation of a septic process.

Cautley<sup>12</sup> (page 130), in writing of suprarenal hæmorrhage, says that probably the most constant cause 'is some form of septic infection, and Riviere<sup>71</sup> suggests intestinal toxæmia as a possible explanation. These several points very strongly suggest that melæna neonatorum is an acute infective micro-organismal disease.

*Lesions found in Cases of Melæna Neonatorum.*—The description of the actual lesion found in these cases is necessarily limited to the fatal and, therefore, the most severe type of case. The stomach and intestines may contain a considerable amount of blood, more or less disorganised in different parts of the canal.

*Conditions present in Stomach and Duodenum.*—W. J. Mayo<sup>57</sup> in discussing hæmorrhage from the stomach and duodenum, describes two kinds of acute ulcers, both of which are probably infective or 'toxic in origin.

1. The acute round ulcer of Creuveillier; in several cases the lesion is a small fissure, 'detected with difficulty.

2. The mucous erosion of Dieulafoy, varying to a greater or less extent in size, and causing the affected mucous membrane to weep blood. Like the acute round ulcer, the mucous erosion is probably infective or toxic in origin. The condition of gastrostaxis described by Hale White as occurring in young women not improbably depends on the formation of these minute pore-like erosions, as distinguished from the usual gastric ulcer. In relation to this *vide* Case 21.

These erosions, according to Adami,<sup>2</sup> take the form of small, irregular, shallow pits, giving the mucous membrane a somewhat moth-eaten appearance; there is no infiltration. He says the more severe forms have been encountered in acute sepsis, and more especially in children. In the case of recent ulcers, there is only exceptionally a marked inflammatory reaction. The non-indurated mucous ulcer is usually located with much diffi-

culty, even if the stomach and duodenum are opened carefully, and search of the whole mucous membrane made. There may be merely an ecchymosis of the mucous membrane of the stomach or duodenum. The ulcer varies in size, and may not be larger than a pin's head (as in Case 18); it may be single, or there may be two or three ulcers present. They are usually superficial, but may extend to the muscular coat and even perforate; frequently the base of the ulcer is covered with adherent blood clots, on removal of which vessels are exposed, branches of the gastro-duodenal artery. The ulcer is limited to the upper portion of the duodenum, and this is the more readily understood when it is remembered that the stomach and first four inches of the duodenum are developed from the same part of the foregut. In four of the twenty cases collected by the writer a pathological condition of the duodenal mucous membrane was found to exist, but this means four out of ten post-mortem examinations, or 40 per cent.

The exact conditions found are described in the notes of cases appended, but briefly the lesion varied from an acute inflammation of the mucous membrane of the duodenum, as in Case 13, where the duodenum also contained blood clot, to a definite ulcer, as in Cases 1 and 17, where the base consisted of peritoneum only. In addition, in two cases out of ten post-mortems, an ulcer of the gastric mucous membrane was found.

We thus get an ulcer of the stomach or duodenum present in no less than 60 per cent. of the cases. When it is remembered that the acute ulcer may be no larger than a pin's head, and is sometimes admittedly difficult to find, the actual proportion of cases where such an ulcer or ulcers exist is, in all probability, considerably greater than 60 per cent.

Dusser,<sup>20</sup> quoted by Holt, found an ulcer of the stomach in nine cases, and of the intestine in four cases out of twenty-four post-mortems.

Moyuihan, in his treatise on duodenal ulcer, enumerates fourteen cases of melæna neonatorum where death occurred within a week after birth, where in each case an ulcer or ulcers existed

in the first part of the duodenum. In two of the cases two ulcers were found, and in one case three occurred.

*Fatty Degeneration of Liver.*—In both the cases which occurred in my own practice, very definite fatty changes were present in the liver, indicating the existence of a septic process.

*Infected cord stump* was present in Cases 2 and 10, and in Case 11 there was subperitoneal hæmorrhage beneath the umbilicus, suggestive of a septic condition in that region.

*Thymus.*—In two cases, 2 and 17, the thymus gland is described as being, in the one case, one-quarter its normal size, and, in the other, almost absent. As the function of the thymus is at present unknown, one cannot draw the inference that the deficiency of the gland tissue favours the onset of hæmorrhage, though, if it may be regarded as a collection of lymphatic tissue, it may influence phagocytosis, and, therefore, its deficiency or absence may act unfavourably in any cases of sepsis neonatorum.

Thus, a survey of the pathological conditions present in melæna neonatorum would lead us to conclude that:—

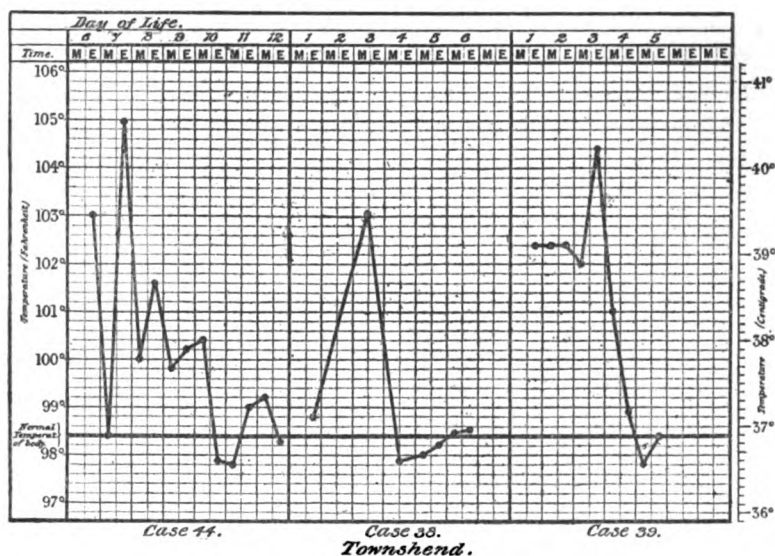
1. The process is due to a toxæmia.
2. The determining organisms are of various kinds.
3. The duodenum plays some special part in the excretion of these toxins.
4. The organisms collect especially in contact with the walls of the blood vessels which become weakened by the action of the toxins, and give way, producing hæmorrhage. In addition to this there is the forcible ejection of an acid chyme through the pylorus tending to bring about a solution of continuity of the mucous membrane of the duodenum.
5. Defective blood coagulation, and syphilis, may in some cases be contributing factors.

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#### CLINICAL FEATURES.

The infant may be to all outward appearances healthy. Frequently within 24—48 hours, even before food has been taken,

an attack of vomiting occurs, and the vomit may, in the first instance, contain a quantity of blood, dark in colour, and altered in character. More frequently the first sign is the passage of a quantity of dark tarry blood, together with the meconium. This is generally repeated after a short interval, and the tendency is for the blood to pass in the form of dark maroon-coloured clots, and if the hæmorrhage persists for it to ooze from the rectum in a thin red stream. The hæmorrhage may quickly



THE TEMPERATURE CHARTS IN THREE CASES OF  
MELENA NEONATORUM.

and spontaneously cease, or it may continue until the infant becomes pale, cold, and collapsed. In this condition the infant frequently dies, practically exsanguinated. There is a rapid loss of weight, diarrhœa is of frequent occurrence, and convulsions may end the scene.

The onset may be associated with pyrexia in the infant (as in Cases 8, 12, 15, 16, and 18), and as exemplified by the temperature charts of Townshend. Holt says fluctuations in tem-

perature during the first three days are so common from disturbances of nutrition that he attaches less importance to this symptom than some writers do. In 14 cases observed by Townshend the temperature of these infants was studied, and in all but two was found to be elevated, at first from  $101^{\circ}$ — $103^{\circ}$ , in one case to  $106^{\circ}$ , and later, on cessation of the hæmorrhages, to sink to normal, often becoming subnormal. The two exceptions to this rule were, in reality, not exceptions, as the temperature was not taken until the second stage of depression was reached.

Melæna is often associated with hæmorrhage from other sources, and Townshend, who gives the following list of sites of hæmorrhage, says the affection is a general and not a local one. "When bleeding occurs in the newborn it is apt to occur from various parts of the economy, the affection being a general and not a local one." The following table gives the various sources of hæmorrhage :—

Intestines	...	...	...	...	20
Stomach	...	...	...	...	14
Mouth	...	...	...	...	14
Nose	...	...	...	...	12
Bowel	...	...	...	...	18
Echymosis of skin	...	...	...	...	21
Scratch of skin	...	...	...	...	1
Cephalhæmatoma	...	...	...	...	3
Meninges	...	...	...	...	4
Abdominal cavity	...	...	...	...	2
Pleural cavity	...	...	...	...	1
Pulmonary	...	...	...	...	1
Thymus gland	...	...	...	...	1
From the gastro-enteric tract, nose, navel, and echymoses	...	...	...	...	3
From the gastro-enteric tract alone	...	...	...	...	19
From the navel alone	...	...	...	...	3
From the echymoses alone	...	...	...	...	6

*Time of Onset.*—Of the 50 cases quoted by Townshend, the bleeding showed itself in all but three within the first seven days of life, the exceptions being on the eighth, ninth, and fourteenth days. The majority of the cases began on the second and third

days, thirteen starting on the second, and sixteen on the third day, while only eight began on the fourth, and two on the first day.


In the Queen Charlotte series of cases, 13 out of the 20 commenced bleeding some time during the second day after birth. Two commenced on the first day, one within half an hour after birth, and the other within 16 hours, two on the third day, and three after the third day.

The examination of the blood does not give very constant results. In estimating blood changes the normal blood count of a newborn infant must be remembered, otherwise a totally false conclusion will be drawn. In the newborn infant both the specific gravity and the percentage of hæmoglobin is at its highest, as is the number of blood corpuscles, the number varying from 4,250,000 to 6,500,000 per c.cm. There is also a greater variation in size in the red cells of newborn children than occurs later. Normoblasts in small numbers occur in the blood of a full term child, but soon disappear.

The leucocytes during the first week vary between 15,000 and 30,000; according to Reider the leucocytes at birth number from 14,200—27,400; second to fourth day, 8,700—12,400; after fourth day, 12,400—14,800. Thus, there is in the newborn a physiological leucocytosis.

The relative percentages of the differing types of white cells are different in young children and adults. Thus: lymphocytes—children 50—60 per cent.; adults 22—25 per cent. Polynuclears less than in adults; adults 70 per cent. Large mononuclear, eosinophile, and mast cells, the same as in adults.

Helmholz, quoted by Moynihan,<sup>61</sup> describes the frequent occurrence of duodenal ulcer in late infancy. A period of weakness, wasting, and anæmia precedes or accompanies the development of an ulcer. Fourteen such cases are reported by Moynihan. Thus, it would appear that both in early and late infancy the existence of a duodenal ulcer must be admitted as a possibility in considering the probability of the cause of any given case of melæna.





## COURSE AND PROGNOSIS.

Of 50 cases of hæmorrhage of the newborn recorded by Townshend 31 died and 19 recovered, a mortality of 62 per cent. He also quotes a mortality of 79 per cent. in 709 cases collected by various authors, probably a high mortality because many mild cases were overlooked. Of the 20 melæna cases collected by me there were 11 deaths and 9 recoveries, a mortality of 55 per cent. Thus, the average mortality is very high. In any case the illness is short and sharp, as the following figures show, and if energetic and prompt treatment be speedily adopted, there is at least a reasonable prospect of favourably influencing the ultimate result. Holt says the duration of the disease in cases which recover is usually one or two days. Townshend says that one-half of the fatal cases lasted one day or less. All the others died within a week except one that died of the effects of the hæmorrhage on the eighth day, or seven days after the bleeding had ceased.

Of the Queen Charlotte Hospital cases, one was fatal in four hours and another in six hours. Of the others, death or recovery usually occurred in two or three days.

Cases where both melæna and hæmatemesis occur appear to be more fatal than those where melæna alone occurs. In eight cases of melæna without hæmatemesis, five recovered and three died, a mortality of 37.5 per cent. In 11 cases of melæna with hæmatemesis four recovered and seven died, a mortality of 63 per cent. Hæmatemesis alone would appear to be a very rare occurrence, being found only once in the Queen Charlotte records. The presence of any one, or more, of the following would imply the existence of a severe type of case, and influence the prognosis unfavourably: A severe and sudden hæmorrhage, profuse, light red, and watery; a purpuric rash on the body and limbs; the co-existence of a high temperature, jaundice, or diarrhœa, indicating sepsis; hæmorrhage into any of the solid abdominal viscera. In fatal cases death results from profound anæmia, collapse, asthenia, and convulsions.

## DIAGNOSIS.

It is necessary to distinguish the condition from a spurious melæna due to blood which has been swallowed either during birth or from a fissured nipple, *e.g.*, Case 4, where an abrasion of the palate was present. Some of the blood in any of these conditions may also be vomited, giving rise to a spurious hæmatemesis. A slight hæmorrhage from the intestine may be easily overlooked. There is usually a slight pink halo on the diaper, surrounding the evacuation, from staining by the blood. Where it is impossible to recognise the corpuscles under the microscope, owing to their disintegration, hæmin crystals may be found as follows: A drop of semi-liquid stool is mixed with a little glacial acetic acid and a few crystals of common salt on a glass slide, and heated to boiling; on drying, dark brown rhombic crystals of hæmin are easily recognised under the microscope if blood is present.

Gastro-intestinal hæmorrhage may be concealed until melæna is noted, or even prove fatal before blood appears in the stools. Thus, any sudden pallor or collapse in a newborn infant should make one suspect the possibility of this condition being present. The date of onset (two or three days), existence of a pyrexia, and association with hæmorrhage elsewhere, are points in favour of a diagnosis of melæna neonatorum. If the bleeding should be chiefly from the nose, suspect syphilis.

*Diagnosis from Hæmophilia.*—True hæmophilia in bleeder families is rarely seen before the end of first year. In melæna neonatorum there is not a family history of bleeding, and such cases are not prone to bleed in after-life.

Dr. Rotch<sup>73</sup> records a case of circumcision being performed a few days after recovery from melæna without any unusual hæmorrhage occurring. In four cases hæmorrhage had occurred from the base of the cord, as well as elsewhere, but the patients recovering, the cords dropped off, one in two days, the other in three or four days after the cessation of the disease, without any fresh hæmorrhage intervening. In melæna neonatorum the pro-

portion of males to females affected is equal. In hæmophilia the proportion of males to females is 13 to 1.

The possibility of an intussusception being present must be considered, the abdomen being carefully examined for the presence of any swelling.

The other infantile diseases, of which melæna is a symptom, are Buhl's Disease, where the hæmorrhages are multiple, and are accompanied by icterus and cyanosis, but no fever, a marked fatty degeneration of the organs being found; and Winckel's Disease, which occurs in institutions in epidemics, when the hæmorrhage is accompanied by icterus and hæmoglobinuria. These cases must also be distinguished from those where the hæmorrhage tends to persist throughout the life of the infant. In 65 cases of congenital narrowing or obliteration of the bile ducts collected by Thomson, hæmorrhages were noted in more than half of the infants who lived more than a few days. Other rare causes of hæmorrhage are cirrhosis of the liver, leukæmia, and pseudo-leukæmia.

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#### TREATMENT.

As Townshend says, if we have in mind that the disease is self-limited we shall not give up a case as hopeless, but will do everything to tide it over the critical period. Treatment may be considered under the following headings :—

- A. Prophylaxis.
- B. Diet, rest, etc.
- C. Drugs.
- D. Injections, hypodermic and rectal.
- E. Serum and vaccine treatment.
- F. Transfusion of blood.
- G. Surgical.

A. *Prophylaxis. Ligature and Treatment of Cord.*—The tape or thread which is to be used for tying the cord should, together with the scissors, be boiled or allowed to soak in some antiseptic solution. After ligature, the cord stump should be

dressed with a powder containing salicylic acid, one part to nineteen parts of starch, and covered with sterile gauze or lint.

*Toilette of Infant's Mouth.*—The infant's mouth should be carefully wiped out with a piece of soft linen or sterile gauze dipped in boiled water. If the dummy teat be used at all, it must be clean.

*Feeding.*—Careful attention must be paid to the condition of the mother's nipples, and suckling from inflamed breasts prohibited. If the infant be artificially fed, all irritating ingesta must be avoided, and careful preparation of the milk, or its substitute, ensured. Attention to these several points will tend to limit the occurrence of a septic condition.

*B. Diet, Rest, etc.*—If hæmorrhage should occur, absolute rest and quiet are necessary. The body and limbs of the infant should be wrapped in cotton wool and surrounded with hot water bottles, or the infant may be put into an incubator. The baby should not be allowed to suck, and small quantities of plain boiled water, or saline solution, may be given out of a spoon or dropper, especially if there be no hæmatemesis. Later, albumen water or whey or alum whey, and subsequently breast milk which has been drawn off.

*C. Drugs.*—It is important to begin treatment at the first indication of bleeding, however apparently insignificant; slight bleeding of the cord may be accompanied by fatal internal hæmorrhage if not stopped immediately. Townshend is sceptical as to the value of astringents, ergot, or mineral acids for internal hæmorrhage. Ergotin, hazeline, calcium lactate or chloride, and bismuth may be given by the mouth, also gelatine with water or whey. With regard to calcium, recent work has cast much doubt on the therapeutic value of calcium in hæmorrhagic conditions (Addis<sup>3</sup>).

In two cases recorded by Schwarz and Ottenburg a small amount of calcium chloride (.1 to 1 per cent.) was added to drops of blood. No coagulation occurred. In these cases, therefore, failure to coagulate was probably not due to lack of calcium. Camphor may be given as a stimulant to counteract the collapse.

It is probably best given hypodermically, the solution being made up as follows: Camphor trit., xii. gr., sp. rectificat, iiss. dr., aq. distillat, ad ss. oz.; v. m. to be injected repeatedly. Adrenalin (1—1000) may be given internally in doses of half to one minim every hour, and may be added to saline solution. In this connection it may be here stated that Rolleston is of opinion that adrenal insufficiency plays some part in setting up a tendency to hæmorrhage in this disease, as it seems to do in others.

It is important not to give any purgatives such as castor oil. Oxygen inhalations may be given at intervals. Allantoin or comfrey root may be tried, as recent successes have been reported in cases of gastric and duodenal hæmorrhage in adults.

*D. Injections. Per rectum.*—Some authors are of opinion that injections into the bowel are to be avoided as tending to set up peristalsis. Astringent injections are practically useless, as the blood comes invariably either from the stomach or from the upper part of the small intestine. A 2—5 per cent. solution of gelatine, or of sterilised salt solution, may be injected per rectum. Adrenalin may be added to the saline.

*Subcutaneously.*—In severe cases a 2 per cent. solution of gelatine in normal saline in doses of 10—15 c.c. every 3—6 hours may be given. It is important to boil the fluid for six hours before use, as it may contain tetanus bacilli; it may give rise to toxic symptoms. Normal salt solution sterilised may be injected with adrenalin.

*Pituitary extract* has proved of such value in cases of collapse that one would certainly give an hypodermic injection of the extract and repeat it if necessary (1/20—1/10 c.c.). Ergotinine citrate, ernutin, or ergot may be injected intramuscularly into the gluteal region.

*E. Serum and Vaccine. Injection of Horse Serum.*—In a case of melæna neonatorum treated by Schwarz and Ottenburg by injection of 20 c.c. of horse serum, no effect was produced, either on the coagulation time or on the hæmorrhages. Many favourable cases have been reported, however. It is important that

the serum should be fresh, as fibrin ferment disappears from serum on standing. How does injection of serum favour coagulation of blood? The fibrin ferment contained in an ordinary dose of serum (10—30 c.c.) is at once neutralised by the antifibrin of the circulating blood. It is possible that the elements of fibrin ferment may be picked out and subsequently used in the production of prothrombin and kinase by the cells which produce these substances. This would afford a rational explanation of serum treatment. E. C. Holt says fresh sterile animal serums, given by the mouth, are successful in arresting various forms of internal hæmorrhage.

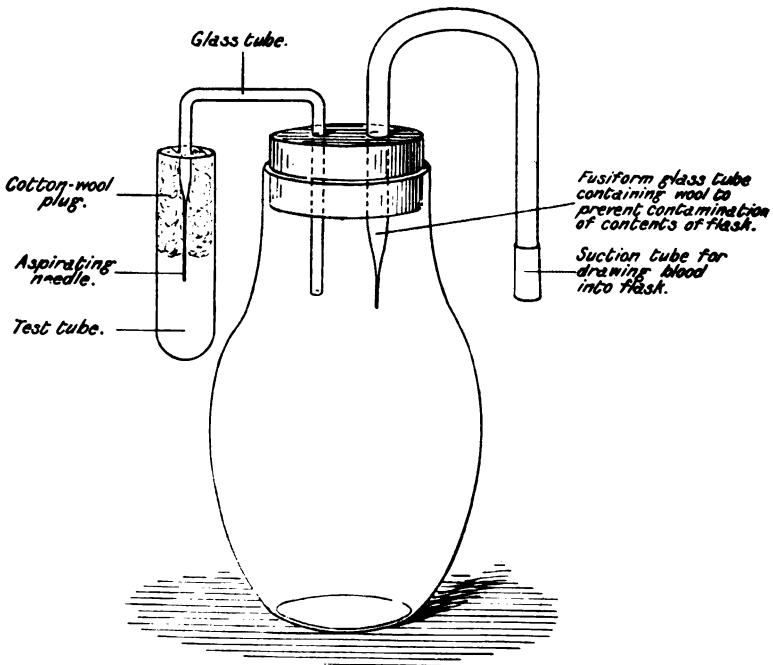
Welch<sup>92</sup> objects to the use of animal serum on the following grounds. It has been shown that large doses of normal horse serum, as well as small ones, will produce anaphylaxis in the guinea-pig. The serum of dogs, sheep, cats, rats, etc., injected into guinea-pigs, followed by second injections with the same serum have produced either severe symptoms or caused death, producing anaphylaxis just as horse serum does.

The principle that the serum of one species of animal is poisonous, and often fatal to a different species, has been well established; therefore, in the treatment of melæna neonatorum Welch<sup>92</sup> uses normal human serum. He gives a record of twelve cases of hæmorrhage in the newborn in which he used normal human serum, all of which have been cured, with eight additional cases, of which seven recovered, the fatal case dying on the fifth day from atelectasis. Welch believes that even if bleeding be due to bacteriæmia, the normal human serum will be still effective. It is now a matter of common knowledge that fresh normal blood serum is often bactericidal; he says it never gives serum sickness or causes anaphylaxis. It may be that the hæmorrhage is partly, and in some cases entirely, controlled by the nutritive effect on the body tissues of the infant; in others, it is possible that a thrombokinase is supplied.

*Dose of Serum.*—Begin with at least 10 c.c. and repeat three times a day if the infant is bleeding only moderately; in severe cases every two hours, and, if necessary, in large quantities. Of

the cases treated by Welch, details of Case 9 are appended, which show the successful result of treatment with normal human serum when the symptoms had been severe.

Case 9, first child; full term; male. Mother had post-partum hæmorrhage. On 4th day, at 4 a.m., the infant had a large hæmorrhage from the bowel and vomited blood-stained fluid. At 4.30 had very profuse hæmorrhage from the bowel and



APPARATUS FOR THE PREPARATION OF NORMAL HUMAN SERUM (WELCH).

vomited a small amount of blood. At 10.30 a.m. bleeding from rectum and small amount of blood vomited. At 11 a.m. hæmorrhage from rectum and small amount of blood vomited. Baby very weak and not able to cry out when injected. Pale and jaundiced; waxy looking. House-physician said that baby had lost half its blood. At 2.15 p.m., small hæmorrhage from rectum. 3 p.m., severe hæmorrhage from rectum. 7 p.m.

small amount of slightly blood-stained small blood clots in the faeces. 10.30 p.m., blood clots in the stools, slight hæmorrhage from nose, mouth, and rectum. During the day 64 c.c. normal human serum injected in six doses. On the 5th day, 8 a.m.: Vomited small amount of blood. 10 a.m., large amount of blood per rectum. This was the last bleeding. On this day 65 c.c. injected in six doses. On the 6th day: 30 c.c. injected in three doses. On the 7th day: 22 c.c. injected in two doses. On the 8th day: 28 c.c. injected in three doses. Total, 209 c.c. in five days.

To prepare normal human serum, insert needle into vein at elbow and withdraw desired amount of blood; allow blood to coagulate in a slanting position in the flask. Withdraw serum as rapidly as it separates, and it is then ready for use. Records of additional cases successfully treated by the injection of normal human blood serum have been recorded by Nicholson and Welch in the Transactions of the College of Physicians of Philadelphia, 1911.

*Injection of Antistreptococcic Serum or Vaccine.*—Injection of 5 c.c. of ordinary stock polyvalent antistreptococcic serum should be given. It certainly appeared to exert a beneficial effect in Case 19. The course of the disease is too short to allow of the preparation of a vaccine from the blood of the patient. As an alternative, a stock vaccine of the bacillus coli communis or other organism found may be injected.

*Antilytic Serum.*—The success of antilytic serum in cases of hæmorrhage from duodenal ulcer in the adult (B.M.J., May 13th, 1911) suggests its exhibition in a case of melæna neonatorum. Such a serum is prepared by Allen & Hanbury, and known as "Antilysin A," which may be given by the mouth in a little water, three times a day, in doses of 1–2 c.c.

The rationale of the treatment is as follows:—Immunity to autolysis or self-digestion is a normal property of healthy cells, and appears to be due to an inhibitory action on the part of its antienzymes. The temporary absence of such antienzymes may, therefore, not only account for the onset of ulceration, but



may cause its continuation. The blood serum of man, horses, etc., has been found to exhibit in a marked degree a resistance to the action of autolytic enzymes. The serum of a normal individual contains stimulins to encourage nutrition and various antienzymes, and they are of the nature of antitrypsin, and may be called antilynsins. This serum is therefore used to raise the immunity of the cells to autolysis.

*F. Transfusion of Blood.*—This was successfully used in a case of hæmorrhage of the newborn recorded by V. Lambert in the *Medical Record* of May 30th, 1908. Transfusion not only replaces the lost blood, but stops the hæmorrhages by supplying new material for the production of fibrin ferment.

Schwarz and Ottenburg record a case of melæna neonatorum where, after failure of injection of horse serum, transfusion of blood was followed by marked temporary benefit, the hæmorrhage ceasing, the coagulation time becoming the same as that of the donor, and the hæmoglobin rising from 30 to 90 per cent. The infant, however, died on the eighth day after transfusion.

Crile has revived transfusion, but even with the improvement of his method sudden death sometimes follows the transfer of blood. Excluding air embolism, death may be due to red cell embolism, hæmolysis, and thrombosis.

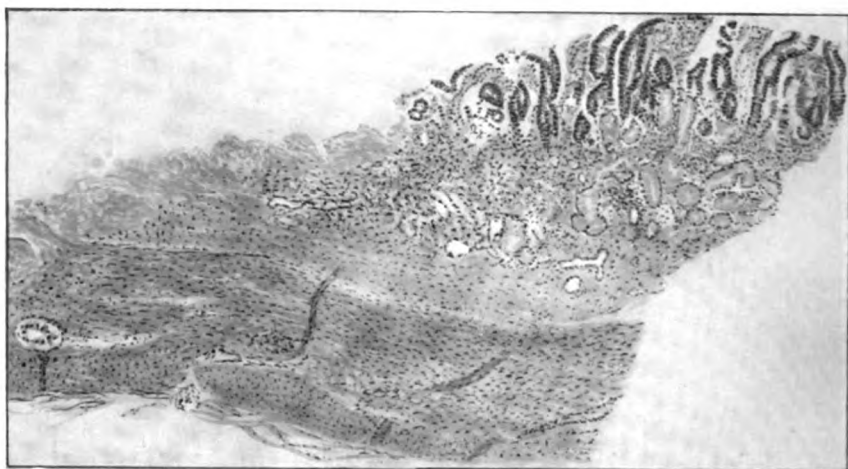
J. G. Hopkins<sup>39</sup> has demonstrated intravascular phagocytosis of red blood cells. This suggests that in transfer of whole blood the cellular elements are just so much foreign material, and that the main virtue lies in the serum alone.

*G. Operative.*—It is impossible to make anything like a reasonable estimate as to the pathological condition which would be found in any given case on opening the abdomen. Duodenal ulcer is present in a sufficiently large percentage of cases to warrant the consideration of operative measures in this condition, and Moynihan says no attempt has yet been made by operation to deal with the condition, but it is quite possible that success would attend such an effort if made early and by expert hands.

The writer would advise attention to prophylaxis, as outlined above, and if melæna does occur, limitation of fluid by mouth to



*The Ætiology of Melæna Neonatorum.*



*M. H. Lapidge.*

× 50.

SECTION A. DUODENAL ULCER. CASE 1.

There is an obvious destruction of the mucous membrane with shelving walls, the base of the ulcer being much smaller in area than the circumference at the edge; the base is covered by a necrotic layer without any evidence of inflammatory reaction.

saline, or boiled water containing adrenalin in sips; external warmth; oxygen at intervals; intramuscular injection of 1/20 c.c. of pituitary extract and camphor to combat shock; subcutaneous saline infusion with adrenalin.

If at hand one of the following:—(a) Antilysin A (Allen & Hanbury), 1 c.c. in water three times daily; (b) antistreptococcic serum, polyvalent, 5—10 c.c.; (c) human serum, which could be prepared after the manner detailed above.

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### NOTES OF CASES.

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CASE 1.—*Melæna. Hæmatemesis, fatal. Duodenal ulcer.*—Mrs. W., three para, was delivered of a full term female child by a midwife on November 23rd, 1910, at 11.30 p.m. The labour was natural; the infant cried lustily at birth, and there was no cyanosis. The two other children were males who have not at any time suffered from hæmorrhage, and are now healthy children. There is no history of any tendency to hæmorrhage in the family of either parent. The infant was put to the breast on the 24th, and went on well till the morning of the 25th, when at 10.30 a.m. she had a severe attack of hæmatemesis followed at 11.30 a.m. by the passage of a quantity of black tarry stools containing blood clot. The amount was too great to have been swallowed in the act of suckling, and there was no crack on the nipples of the mother's breasts, and no bleeding point in the mouth of the infant. I saw the child soon after the first attack of melæna, and found it pale and cold. Adrenalin, v. m. (1—1000) every four hours, and calcium chloride, 2 gr., every two hours, were given by the mouth, and the child was wrapped in cotton wool. The passage of a quantity of blood from the bowel recurred at 12.30, 4, and 8.30 p.m. on the same day, the blood being now redder in character. On the 26th there was a further attack of melæna in the early morning; the baby was by this time very collapsed, and did not respond to saline injections. No further hæmorrhage took place, but the baby died at 7.30 a.m. on the 27th November, about eighty hours after birth, and forty-five hours after the onset of the hæmatemesis. Only a partial post-mortem was allowed, and this was made about nine hours after death in the afternoon of November 27th.

*Post-mortem.*—The body was extremely pallid, and on opening the abdomen the tissues and organs were very pale. There was no naked-eye evidence of sepsis of the cord, and there were no cutaneous hæmorrhages. There was a quantity of dark blood in the stomach and intestine, extending into the colon. On the posterior and lower surface of the duodenum, about half an inch from the pylorus, was an ulcer about a quarter of an inch in length with steep edges; this was filled with blood clot which had to be washed away with a stream of water to expose the base, which consisted merely of peritoneal covering. In stretching the

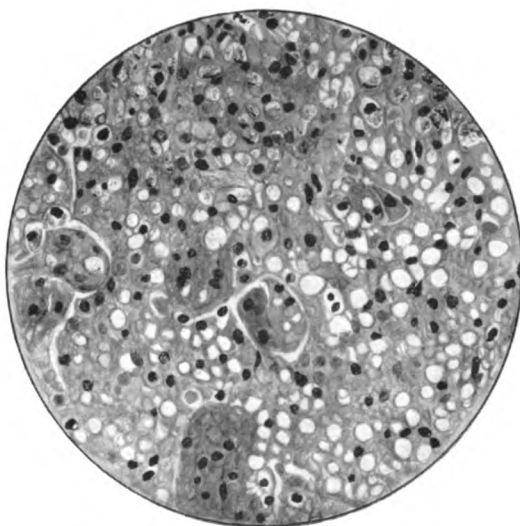
bowel to see the ulcer to better advantage, a tiny hole was made in the peritoneal base. The lungs and thymus appeared healthy. The following organs were removed and examined subsequently, and the conditions found were as follows: *The heart* shows no abnormalities of the valves or general naked-eye pathological changes. *The liver* presents patches of necrosis of the hepatic cells in which the cell protoplasm is cloudy, the nuclei stain badly, and the capillary vessels are, to some extent, obliterated by swelling of the cells. This change is suggestive of a toxæmic condition. *The spleen* is healthy. *The umbilical cord* shows no change suggestive of a septic infection via the umbilicus. There is no evidence of *sypilis* in any of the organs. *The stomach* itself presents no ulcer discoverable by the naked eye. *The duodenum*: Careful examination of sections made from the duodenal ulcer leaves the question of its origin doubtful; there is an obvious destruction of the mucous membrane, with shelving walls, the base of the ulcer being much smaller in area than the circumference at the edge; the base is covered by a necrotic layer without any evidence of inflammatory reaction. (It is even possible that the ulcer may be an artefact.) *Intestines*: Sections made from the intestines show that it is filled with debris of epithelium, blood, and mucoid material; there is no ulceration discoverable, and this material is the result of post-mortem changes, except in the case of the blood. *Kidneys and suprarenals* healthy; the latter do not show any great vascularity or medullary hæmorrhages.

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CASE 2.—*Melæna. Hæmatemesis, fatal. Infected cord. Bacteriæmia.*—Mrs. D., seven para, aged 31, was delivered by a midwife of a full term male child on the evening of the 19th March, 1911. The labour was normal; the baby was a good colour, and cried strongly. None of the other children suffered from hæmorrhage, and there is no "bleeder history" in the family of either parent. The infant was put to the breast on the following day, and nothing untoward occurred. Late in the day of March 21st there was slight vomiting of "brown coffee-coloured" fluid. On the morning of the 22nd the baby was found with the diapers soaked in blood. No further vomiting. Each diaper taken off during the day was soaked in blood. The baby was seen by my assistant in the evening; there had been no hæmorrhage from the cord. Calcium chloride was given by the mouth every four hours, and retained. I saw the baby on the morning of the 23rd and found it very pale and collapsed. Blood was passed in clots per anum at intervals. Adrenalin x. m. in saline 5 ounces, was injected under the skin. The child was so exsanguinated that no blood could be obtained for examination by deeply pricking the ear or finger. *Melæna* occurred at intervals during the day, and the child died at 6 p.m., about ninety-six hours after birth, and fifty-six hours after the onset of hæmatemesis.

*Post-mortem* made the same evening about three hours after death; no examination of the brain being allowed. The tissues were very pallid; the thoracic and abdominal organs were removed, a specimen of blood being previously withdrawn from the vena cava by means of a sterilised syringe, also a specimen of pericardial fluid, as on opening the pericardial

*The Ætiology of Melæna Neonatorum.*



*M. H. Lapidge.*

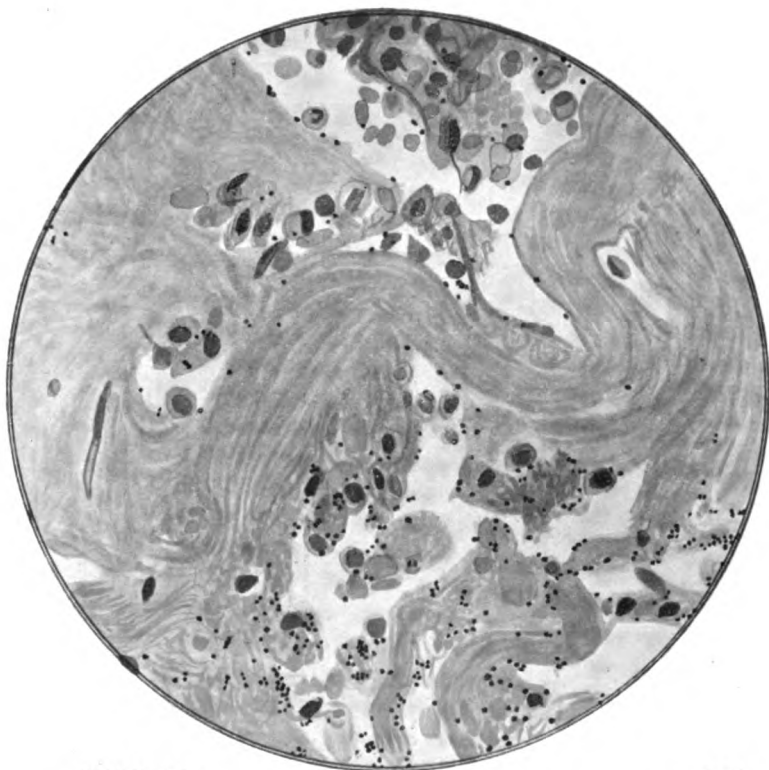
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SECTION B. FATTY DEGENERATION OF LIVER. CASE 2.

The liver shows marked fatty degeneration of the hepatic cells; there is no increase of fibrous tissue; no evidence of syphilis, and no necrosis taking place.



*The Ætiology of Melæna Neonatorum.*



*M. H. Lapidge.*

× 600.

SECTION C. UMBILICAL CORD STUMP. CASE 2.

The cord is thickly infiltrated by leucocytes at the base, and has all the appearance of a lesion which is the result of infection; there are many gram-positive cocci in the tissues, large masses of them being grouped in places.





sac the fluid was found to be in excess. No lymph was visible on the inner side of the pericardium. The fluids and viscera were subsequently examined, and the following conditions were found to be present: *The stomach and intestines*, when cut open, contained altered blood throughout their length with few exceptions. The most careful examination showed no evidence of an ulcer in either stomach or intestine. Microscopically, the intestinal membrane and gastric mucous membrane were well preserved and showed no obvious pathological change. *The liver* showed marked fatty degeneration of the hepatic cells; there was no increase of fibrous tissue; no evidence of pyphilis; and no necrosis taking place. *The kidneys* showed no obvious pathological change. *The spleen* showed no change. *The suprarenal capsules* showed the usual distinction between the cortex and medulla; the cells appeared normal, and there was no hæmorrhage nor dilatation of vessels in them. *The umbilical cord stump* was abnormal. It was thickly infiltrated by leucocytes at the base, and had all the appearance of a lesion which was the result of infection; there were many gram-positive cocci in the tissues, large masses of them being grouped in places. As regards organisms in the other tissues, occasional gram-positive rods were seen on the surface of the liver and intestines, which might be post-mortem effects. *The blood from the vena cava* was shown to contain many gram-negative bacilli which had the cultural characters of bacillus coli, and in subculture it was found that there were also present a very few colonies of the staphylococcus aureus and the streptococcus faecalis. *The pericardial fluid* was shown to contain a small number of micrococci and many gram-negative bacilli, which showed the cultural characters of the bacillus coli; the micrococci were streptococci of the faecalis type, and a few staphylococci of the albus type. The bacillus, isolated, had the characters of the bacillus coli and not those of Gärtner's bacillus. The presence of the bacilli may be variously explained as due to (1) Post-mortem contamination; (2) post-mortem invasion of the tissues (post-mortem made three hours after death); (3) ante-mortem presence and being the causal organism.

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The following cases are taken from the Queen Charlotte's Hospital Reports:—

CASE 3.—*Melæna. Hæmatemesis. Recovery.*—Primipara delivered November 4th, 1904, 7 p.m. Full term; natural labour. Male child, 5 lbs. 10 ozs. About forty-eight hours later the baby assumed a grey colour, and had a whining and painful cry. Shortly afterwards passed a small tarry motion and brought up a small quantity of altered blood; cried continually as if in pain. The following mixture was given: Tr. ferri perchloridi, ii. m., pot. brom., 1 gr., liq. calc. chlorid., v. m., glycerine, v. m., aqua ad  $\frac{1}{2}$  drachm, o.h. No further hæmatemesis or melæna occurred; improvement continued, and the patient appeared quite well on the 16th November.

CASE 4.—*Melæna. Abrasion of palate. Recovery.*—Two para, delivered June 5th, 1904, 3 a.m. Normal labour. Full term male child, 6 lbs. 6 ozs. On the 12th June a slight bleeding occurred from a superficial abrasion

in the palate, and the child vomited altered blood. Two days later tarry motions were passed, and the melæna continued till June 16th, when a local application of cotarnin hyd. 1/16 gr. temporarily stopped the bleeding. Some extravasation of blood now occurred under the skin of the back. The next day there was further bleeding, and adrenalin chloride 1 in 1000 was applied locally, liq. calc. chlor., v. m., being given internally every two hours. On the 19th the motions were still tarry. There was no further melæna after the 21st, and the baby gradually improved in colour and gained in weight, iron being given internally in minim doses. There was profuse desquamation of the surface of the soles of feet and tips of fingers. The child was fed with mother's milk from a spoon. The cause of the abrasion of the palate is not mentioned; the extravasation of blood under the skin of the back would suggest that intestinal hæmorrhage was also occurring.

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CASE 5.—Melæna. Recovery.—Primipara, delivered December 18th, 1902, at 11.15 p.m. Full term. Normal labour. The infant passed blood per rectum during the night of the 22nd December, and also during the whole of the 23rd. The child, who had previously taken the breast well, was now put on albumen water, 1 oz. every two hours, and given a mixture containing ext. ergot, v. m., tr. camph. co., x. m., and calcium chloride. v. gr. There was no further melæna after this day, and the baby was put back to the breast two days later. This was evidently a mild case, as the quantity of blood lost is noted as not having exceeded 3 ounces.

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CASE 6.—Melæna. Recovery.—Two para, confined September 8th, 1.45 a.m. Normal labour. Full term female child weighing 7 lbs. 10½ ozs. About thirty hours after birth passed a quantity of blood per rectum, partly red, but for the most part "tarry" in character; a similar evacuation occurred three hours later. Given liq. calc. chlor., ii. m., with tr. ferri perchlor., i. m. every two hours. The next day there were six such evacuations, the baby now being blanched, but took well (breast milk drawn off). On the 12th September the general condition was good, and no further hæmorrhage occurred, and a week later the baby had put on 12 ounces in weight, and was doing well, though still very pale. This was evidently a severe case, and the recovery is noteworthy.

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CASE 7.—Hæmatemesis and melæna. Fatal. ? Gastric ulcer.—Primipara delivered on December 19th, 1905, at 10 a.m., of a full term female child, weighing 7 lbs. 4½ ozs. Early on the second day, hæmatemesis occurred. Lactate of calcium, 2 gr., every four hours was given. More profuse bleeding occurred on the 3rd day, which temporarily ceased after giving 30 minims of adrenalin (1 in 3000). Further hæmatemesis accompanied by melæna took place; 20 grains of calcium lactate and 5 grains of gallic acid were given, but the passage of blood per rectum remained uncontrolled, and the infant died four days after birth.

*Post-mortem.*—Bright red blood in patches throughout the intestines. Stomach full of altered blood, a few bright red patches in the mucous

membrane, and in one place a perforation (post-mortem digestion?) a few petechial hæmorrhages occurred on the left pleura, and all the organs were very anæmic. The puerperium of the mother was normal.

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CASE 8.—*Melæna. Fatal.*—Three para, delivered of a full term male child weighing 9 lbs., on July 1st, 1906, at 2 p.m. There was considerable post-partum hæmorrhage, which also occurred in the two previous confinements. On the eighth day after delivery the motion contained dark altered blood, and a more severe hæmorrhage occurred later in the day; the infant's temperature at the time of onset of the hæmorrhage was 102° F. The baby was pale, but took the breast well. The next day the temperature was normal, and the baby seemed better, but on the 11th breathing was difficult in the morning, and more blood was passed per rectum. Brandy and oxygen were administered, but the baby became weaker in the afternoon, with rapid respiration and marked pallor, and death occurred. No post-mortem was allowed. There was no maternal sepsis.

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CASE 9.—*Melæna. Recovery.*—Three para, delivered of a full term female infant weighing 6 lbs. 15 ozs., on the 23rd June, 1908. Normal labour. On the second day the infant had two attacks of melæna; no tumour was felt in the abdomen. There was no special treatment, and the hæmorrhage ceased in less than twenty-four hours.

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CASE 10.—*Fatal hæmorrhage from the cord. ? Syphilis. ? Sepsis. ? Hæmophilia.*—Two para, delivered of a full term male child weighing 6 lbs. 11½ ozs., on August 12th, 1907, at 5.30 a.m. Normal labour. At 2 a.m. on August 14th the cord stump was found to be bleeding, and as this did not yield to firm pressure, the cord was removed with scissors, and the umbilicus under-run with a suture. The cord was noticed to be soft and pus-like. Bleeding recurred again at night. The umbilicus was excised, the hypogastric under-run, and the wound stitched up. On the 15th oozing of blood still taking place; stitch holes, after removal of stitches, were bleeding. The cautery was freely used, and as adrenalin had no effect, the wound was dressed with tr. ferri perchlor., and the cicatrix filled with powdered calcium chloride, two grains of this latter drug being also given internally every three hours. The child was very blanched on the 16th, and although it was placed in an incubator and infused with saline, it died soon after mid-day.

*Post-mortem, August 17th.—Thorax:* All organs anæmic. Thymus about one quarter its natural size. *Abdomen:* Contained about 3 ozs. of dark blood clot; adherent blood clot on left side of abdominal wall in renal region. *Subperitoneal* ecchymosis extending over area size of a five-shilling piece beneath umbilicus. *Suprarenals* healthy. *Spleen* enlarged and hard. *Liver* hard; not enlarged. *Retropertitoneal* tissue thickened. ? Congenital syphilis. Here the cord was noted to be soft and rather pus-like, suggesting sepsis, whilst the hardening of liver and spleen and

thickened retroperitoneal tissue suggested that syphilis might also be a contributing cause. A suggestion as to hæmophilia being the cause was also made.

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CASE 11.—*Melæna neonatorum*. Fatal.—Mother, aged 16; primipara. Confined October 8th, 1908, 7 p.m. Normal labour. Female infant, 5 lbs. 1½ ozs. in weight, 19½ inches long. Full term. Half an hour after birth the infant began to bleed from the rectum, and became markedly anæmic. A saline infusion (vi. ozs.) with adrenalin v. m. was given per rectum. The hæmorrhage stopped for an hour or two, but recurred. Another saline injection was given, but the child never rallied, and died at 5 a.m., ten hours after birth. The *placenta* was relatively large, and weighed one quarter that of the child; it was fibroid, and displayed generally the features ascribed to a syphilitic placenta. No mention of syphilis in the mother. The puerperium was normal.

*Post-mortem*, October 9th.—*Thorax*: Heart contracted and empty. *Abdomen*: Slight subperitoneal hæmorrhage beneath umbilicus. Small intestine full of blood. No other obvious disease. Stomach and rectum contracted and empty. The condition of the placenta would here suggest syphilis as a possible contributing factor. Note also the subperitoneal hæmorrhage beneath the umbilicus.

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CASE 12.—*Melæna*. Recovery.—Two para, delivered May 30th, 1909, of a full term female child weighing 6 lbs. 12½ ozs. Normal labour. On the second day baby vomited "coffee grounds" once and passed tarry motions. Lost 9 ozs. in weight since the previous day; there was no abdominal tumour. There was no recurrence of the hæmorrhage, and the infant again rapidly gained weight. The baby's temperature was 99° F. at the onset of the hæmorrhage, having previously been subnormal; it returned to normal in forty-eight hours. The pyrexia at the time of onset of hæmorrhage is noteworthy in this case.

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CASE 13.—*Hæmatemesis and melæna*. Fatal. Acute duodenitis.—Primipara, delivered December 1st, 1909, at 6.40 p.m., of a full term male infant weighing 5 lbs. 15 ozs. Forceps delivery. About forty hours after birth the baby suddenly vomited about 6 drachms of bright blood and passed some dark blood per rectum. The child was pale and cold. No lump in abdomen. Brandy and ernutin, one minim of each, was given every three hours and saline per rectum. On the next day there was bright blood passing per rectum; a starch and opium enema was given. During the night there was further hæmatemesis on three occasions, but no melæna, and the infant died on the morning of December 5th.

*Post-mortem*.—The duodenum below opening of bile duct contained clotted blood. The mucous membranes of the duodenum were acutely inflamed; the inflammation became less marked in the jejunum, but most of the small intestine contained altered blood, which apparently came from the duodenum. The pancreas and adrenals were normal. The

umbilical artery and vein were normal; the kidneys pale, but healthy; the brain pale, but normal; the lungs well expanded; and the heart normal. In this case, the acute inflammation of the mucous membrane of the duodenum and the contained blood clot are to be especially noted.

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CASE 14.—*Melæna*. Recovery.—Primipara, delivered March 30th, 1910, at 8 p.m., of a full term female infant weighing 8 lbs. 1 oz. Normal labour. On April 1st blood was passed in the stools, and later in the day it was bright red. Child obviously in pain. Good deal of tympanites. No suggestion of intussusception. Child breast-fed. Mother's left breast tender, and she is also slightly sapræmic. No infantile pyrexia. There was no recurrence of the hæmorrhage, and no special treatment was adopted. This was apparently a very mild case.

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CASE 15.—*Melæna*. Recovery.—Primipara, delivered May 9th, 1910, at 8.50 p.m., of a male child, 4 lbs. 8½ ozs. Slight ante-partum hæmorrhage. About twenty-four hours after birth the infant passed two large dark "tarry" motions. No pain or vomiting. Abdomen normal. There were three further similar evacuations of blood on the following day, but this did not again recur, and the infant made a good recovery. The infant had a temperature of 100·8° F., and the mother had sapræmia with temperature of 100·8° F.

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CASE 16.—*Melæna*. Fatal. Duodenal ulcer.—Primipara, delivered on September 29th, 1910, at 10.15 a.m., of a full term female infant, 6 lbs. 6 ozs.; labour normal. When forty-eight hours old the baby passed blood in the stools, dark and "tarry" in character. There was no cyanosis and no jaundice. Urine normal in colour. The infant rapidly became pale, with rapid pulse and dyspnœa, and within four hours was dead. Treatment consisted in giving sips of brandy and water. At the time of onset of the hæmorrhage the baby's temperature was 100·4° F. There was no maternal pyrexia.

*Post-mortem*.—The rectum contained partly digested blood. Colon and small intestine full of dark blood, which became brighter as the duodenum was approached. Stomach greatly distended with air; contained a little mucus and a few curds; no blood. Duodenum: Half an inch beyond the pyloric sphincter, on the posterior wall, was an ulcer whose base was formed by peritoneum and whose walls were sharply cut, three lines in diameter; no vessel seen eroded, but blood was bright coloured; peritoneum normal.

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CASE 17.—*Melæna*. Hæmatemesis; fatal.—Two para, delivered on October 2nd, 1910, at 8.50 a.m., normal labour. Male infant, 6 lbs. 15½ ozs. The infant was fed with difficulty, and seldom cried. On the third day it appeared very ill, having passed some dark "tarry" stools during the night. Vomiting of dark red blood now occurred, and the extremities became cold. One grain calcium lactate was given every

hour, but the child died within twenty-four hours of the onset of the hæmorrhage. There was no temperature in mother or infant.

*Post-mortem.*—Marked pallor. Heart, lungs, trachea, and œsophagus normal. *Thymus almost absent.* No obvious lesion in abdominal viscera.

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CASE 18.—Melæna. Hæmatemesis; fatal. Duodenal ulcer.—Nine para, delivered on October 19th, 1910, at 11.55 p.m.; normal labour. Female infant weighing 7 lbs. 4½ ozs. About twenty-five hours after delivery the baby became pale and vomited bright red blood with one or two small clots. Two hours later melæna commenced. A cylindrical clot, corresponding in outline to lumen of small bowel, was passed. Further melæna occurred, and the baby became very pale and thirsty. Given adrenalin xx. m., with saline solution to a drachm, every two hours. At mid-day 1/20 c.c. of pituitary extract with 10 c.c. of saline was injected, and half an ounce of albumen water given every hour by the mouth. During the afternoon and evening adrenalin, ii. m., was injected into the gluteal region. Pituitary extract was again injected, and oxygen inhalations were given. Although there was no further melæna after 5 p.m. on the 21st the pallor increased, and the baby died at 4 a.m. on the 22nd October. There were no purpuric spots, and no signs of hæmorrhage from any other source. The baby's temperature rose to 99° F. on the third day from 98° F. on the second. No maternal sepsis.

*Post-mortem.*—Great pallor of tissues. *Lungs and heart normal.* A small patch of ulceration in the second part of the *duodenum*, extending down to the muscular coat and of the size of a large pin's head; the rest of the intestine was congested, but no other abnormality was found. A blood culture was negative.

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CASE 19.—Melæna. Hæmatemesis. Recovery.—Primipara, delivered of a full term female child on November 2nd, 1910, at 1.20 a.m., 6 lbs. 3½ ozs. Forty-six hours after birth, and one and a half hours after going to the breast, the baby gave a hoarse cry and immediately vomited about a teaspoonful of bright blood-stained fluid. A few small clots were seen, also a few "coffee grounds." No other focus of hæmorrhage. Given 2 grains of calcium lactate every two hours and a teaspoonful of albumen water every hour. On November 4th 5 minims of adrenalin were added to the albumen water, and 5 c.c. of antistreptococcic serum were injected with saline per rectum, half of which was retained. In the early morning melæna occurred. Later in the day the infant was given breast milk. A blood count resulted as follows: Red corpuscles, 7½ millions; hæmoglobin, 95 per cent.; leucocytes, 11,000. Polymorphonuclear, 51 per cent.; lymphocytes, 40 per cent.; transitional, 5 per cent.; Eosinophile, 4 per cent.; coagulation time, 3 minutes. On the 6th November 5 c.c. of serum again injected. One stool showed altered blood, but afterwards there was no recurrence of hæmorrhage, and the child left the hospital well on the 16th November. A blood culture showed presence of non-gram-staining rods, probably bacillus coli. Altogether 10 c.c. of serum was given, of which about half was retained.

CASE 20.—Melæna. Hæmatemesis; fatal. Gastric ulcer.—The following notes are recorded by Dr. W. B. Ray of Harrogate in the *British Medical Journal* of August 5th, 1911: An apparently healthy full time male child was seized about sixteen hours after birth with violent and repeated attacks of hæmatemesis, bright red in colour. This was accompanied later by the passage of dark red blood by the bowel. The shock was too great, and the child died about six hours after.

*Post-mortem.*—An ulcer about the size of a sixpence was found in the pylorus. Dr. Ray has kindly provided me with the following additional note: The ulcer was on the gastric side of the pylorus; it involved the circular muscular fibres, forming the pyloric sphincter; the rugæ of the gastric mucous membrane were more or less eaten away at this spot; one could easily put the tip of the finger into the erosion.

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CASE 21.—Multiple hæmorrhages, especially in stomach, duodenum, jejunum, ileum, and lung.—Eliza Smith, 1½ days old. The only history in this case was that the patient had been born upon April 20th, and had suffered from hæmorrhage from the mouth and rectum; she was admitted to Guy's Hospital upon April 21st, and was infused with saline, but died early the following morning at 4 a.m. The following are the post-mortem notes made by Dr. Herbert French:—

"The body of the infant did not seem abnormal externally; the umbilical cord was well tied and perfectly healthy in itself. On opening the body one was at once struck by the fact that the stomach, duodenum, jejunum, and ileum were of a black colour externally owing to the thinness of their wall, allowing one to see the dark blood clot within them, whilst in marked contrast the cæcum, ascending colon, transverse colon, descending colon, sigmoid, and rectum were perfectly white and contained no blood. On opening up the alimentary canal it was found that the whole of the stomach and small intestines contained, as it were, a cast of clotted blood, but no single bleeding point could be detected. The blood which had been passed per rectum did not seem to have been derived from the colon itself, but rather to have been derived from the small intestines, and to have been discharged per rectum as soon as it reached the colon. Similarly, the œsophagus contained no blood, so that that which had been vomited was probably derived from the stomach. One knows of these cases quite well, but their explanation is obscure. The lungs in this patient contained numerous hæmorrhagic foci; these may have been due to actual hæmorrhage in the lung itself, but quite as possibly the child may have inhaled blood that was in process of being evacuated upwards from the stomach. There were no other hæmorrhages to be found in any of the organs, and there was no purpura. The condition seemed to be allied to that which in older people produces severe hæmatemesis by what Dr. Hale White calls gastrostaxis. When the blood clot was washed away from within the alimentary canal, there was no indication of any inflammation. The pyramids of the kidneys presented a very beautiful picture of streaky deposits of uric acid in the tubules opening into the calyces."

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No. of Case.	Sex of Infant.	No. of Confinements.	Time of onset of Hæmorrhage.	Result. Recovery or Death.	Melæna or Hæmatemesis	Sepsis in Mother or Infant.	Lesions present.	Character of Labour.
1	Female	3	35 hours after birth	Death in 80 hours after birth	Hæmatemesis and melæna	Maternal sapræmia	Duodenal ulcer. Fatty liver	Normal
2	Male	7	Late in the 2nd day	Death in 96 hours after birth	Hæmatemesis and melæna	Infected cord stump	Bacteriæmia. Marked fatty changes in liver	Normal
3	Male	1	48 hours	Recovery within 24 hours	Hæmatemesis and melæna	No record		Normal
4	Male	2	7 days	Recovery in 5 days	Hæmatemesis and melæna	No record	Ulcer on palate. Subcutaneous extravasation of blood on back	Normal
5	Not stated	1	4th day	Recovery in 1 day	Melæna	No record		Normal
6	Female	2	Early on 2nd day	Recovery in 2 days	Melæna	No record		Normal
7	Female	1	Early on 2nd day	Death on 4th day	Hæmatemesis and melæna	No maternal sepsis	? Perforated gastric ulcer	Normal
8	Male	3	8th day	Death in 2 days after onset	Melæna	No maternal sepsis. Infantile pyrexia	No post-mortem	Normal. Post-partum hæmorrhage

No. of Case.	Sex of Infant.	No. of Confinements.	Time of onset of Hæmorrhage.	RESULT. Recovery or Death.	Melena or Hæmatemesis.	Sepsis in Mother or Infant.	Lesions present.	Character of Labour.
9	Female	3	2nd day	Recovery in less than 2 hours	Melena	No record		Normal
10	Male	2	48 hours	Death 24 days after onset	Hæmorrhage from cord	No record	Cord soft and pus-like. Spleen and liver hard. ? Syphilis. Thymus only quarter normal size	Normal
11	Female	1	Half an-hour after birth	Death in 10 hours after birth	Melena	Puerperium normal	Subperitoneal hæmorrhage beneath umbilicus. Small intestine full of blood. Placenta (?) syphilitic	Normal
12	Female	2	2nd day	Recovery in 24 hours	Hæmatemesis and melena	Infantile pyrexia		Normal
13	Male	1	50 hours after birth	Death on 4th day after birth	Hæmatemesis and melena	None	Mucous membrane of duodenum acutely inflamed. Blood clot present in duodenum.	Forceps delivery
14	Female	1	2nd day	Recovery within 24 hours	Melena	Sapremia in mother with tender breast. No infantile pyrexia		Normal

No. of Case.	Sex of Infant.	No. of Confinements.	Time of onset of Hemorrhage.	Result. Recovery or Death.	Melena or Hæmatemesis.	Sepsis in Mother or Infant.	Lesions present.	Character of Labour.
15	Male	1	24 hours	Recovery within 48 hours	Melena	Infantile pyrexia. Sepsis in mother		Slight ante-partum hæmorrhage, otherwise normal
16	Female	1	48 hours	Death in 4 hours after onset of hæmorrhage	Melena	Infantile pyrexia. No maternal pyrexia.	Ulcer of duodenum half-an-inch beyond pylorus	Normal
17	Male	2	3rd day	Death about 24 hours after onset of hæmorrhage	Hæmatemesis and melena	No pyrexia in mother or infant	Thymus gland almost absent	Normal
18	Female	9	25 hours	Death in 28 hours after onset of hæmorrhage	Hæmatemesis and melena	Infantile pyrexia	Ulcer of duodenum. Blood culture was negative	Normal
19	Female	1	46 hours after birth	Recovery after 4 days	Hæmatemesis and melena	Not mentioned	Blood culture showed presence of B.C.C. Coagulation time 3 minutes	Normal
20	Male	—	16 hours after birth	Death in 6 hours after onset	Hæmatemesis and melena	No record	Gastric ulcer	Normal

## APPENDIX.

The following are the details of the Queen Charlotte Hospital cases:—

Year.	No. of Births.			No. of Deaths of Children.
1896	...	...	1,118	71
1897	...	...	1,077	58
1898	...	...	1,052	88
1899	...	...	1,147	58
1900	...	...	1,176	82
1901	...	...	1,263	93
1902	...	...	1,281	79
1903	...	...	1,465	80
1904	...	...	1,355	93
1905	...	...	1,560	95
1906	...	...	1,738	114
1907	...	...	1,696	95
1908	...	...	1,723	122
1909	...	...	1,704	117
1910	...	...	1,764	113
Total	...	...	21,119	1,358
Cases of Melæna Neonatorum	...	...	...	17
Recovered	...	...	...	9
Died	...	...	...	8

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*A Case of Traumatic Aneurysm of the Heart.*

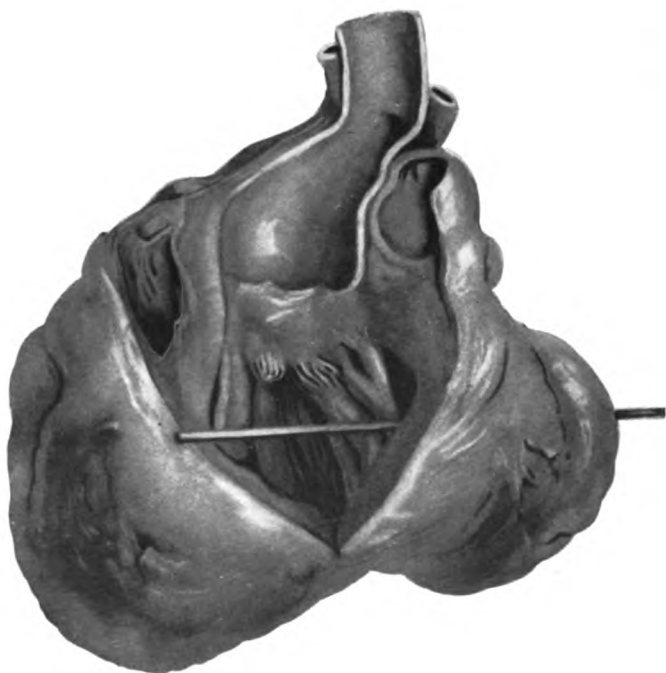


FIG. I.—Traumatic Aneurysm of the left ventricle. The probe passes from within the left ventricle through the aneurysm and projects from the latter at its site of rupture.

# A CASE OF TRAUMATIC ANEURYSM OF THE HEART

OF A CHILD AGED THREE RESULTING FROM A FALL  
FROM A THIRD STOREY WINDOW TO THE GROUND,  
PRODUCING NO DEFINITE CARDIAC SYMPTOMS DURING  
LIFE, BUT LEADING TO SUDDEN DEATH BY  
SPONTANEOUS RUPTURE ON THE TWENTIETH DAY  
AFTER THE ACCIDENT.

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By

HERBERT FRENCH.

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THE specimen is illustrated in Fig. I., which shows the heart opened along the front of the left ventricle, with a probe passed from the interior and projecting through a small spontaneous rupture in the aneurysm, which was itself about one and a half inches in diameter.

The patient was a well-developed little girl three years of age, who on the 17th August, 1911, fell from a third storey window to the ground. She was picked up unconscious and brought to the hospital, where she was found to have an axillary temperature of 97° F., a pulse rate of 140, and a fractured left femur, but no other obvious ill-effects of the fall. There was no fracture of the skull. The symptoms of her concussion passed off during the next day or two; on August 22nd a general anæsthetic was given, and Mr. Arbuthnot Lane operated and fixed the fractured ends of the broken left femur together in



perfect position by means of long, narrow metal plates and screws. The little girl took the anaesthetic well, and thereafter seemed to be progressing favourably, though it was noted that the pulse rate was persistently rapid. On September 3rd the patient had some kind of an attack which was described by the nurse as a "fit," but no very definite details of this are available; it was quite transient and, at the time, did not seem to be important. On the evening of September 5th another convulsive seizure occurred, and this began definitely in the right hand, spreading rapidly up the right arm, and thence over the whole body, whilst at the same time there was internal strabismus of the right eye. The attack lasted only a few minutes, and after it the child seemed to be quite well, though, in view of the cardiac lesion found post-mortem, it seems likely that the seizure was due to a small cerebral embolism. In the evening of September 5th the child appeared to be quite well again when she suddenly sat up in bed, gasped for breath, and fell back moribund, dying within two minutes. From the post-mortem findings this terminal attack was probably synchronous with the rupture of the traumatic cardiac aneurysm into the pericardium.

At the autopsy next day the operation site was found to be perfectly healthy, and all deformity at the site of fracture had been corrected by the plates.

The brain weighed 1,030 grams and it presented no naked-eye abnormality, no local softening, no hæmorrhage, and no obvious embolus in any vessel, though there probably was a small embolus which was not discovered on naked-eye examination. There was no affection of any of the cerebral sinuses nor any fracture of the calvarium or its base.

On opening the thorax the lungs looked natural, but the pericardium was acutely distended with what proved to be blood, part of which was clotted and part still fluid. The total amount of this fluid blood that could be collected and measured was seven ounces. Bulging from the left side of the left ventricle, immediately below the auriculo-ventricular junction, and oc-

cupying rather more than half the distance between this auriculo-ventricular junction and the apex of the heart, there was a typical globular aneurysm, very much thinner walled than the rest of the heart, apparently of quite recent date, and partly adherent by recent clotted blood and fibrin to the corresponding area of the parietal pericardium. In the centre of the bulge, and rather like the opening there is at the apex of a mature puff-ball, there was a small aperture, about three millimetres in diameter, through which blood had escaped from the interior of the heart through the cardiac aneurysm into the pericardium. One's impression of the aneurysm was that it had come about from softening of the heart muscle as the result of severe contusion and bruising at the time of the fall from the third storey window twenty days before death. The sequence of events had been, I think: bruising of the heart muscle as the result of the sudden excessive jerk it got when the patient struck the ground, together with some hæmorrhage into the heart muscle at the site of the bruising; consequent softening and weakening of the affected part of the ventricular wall so that it became bulged out more and more by the pressure of the blood within the ventricle during systole; until, from the stretching and thinning, rupture ultimately took place at the thinnest part. To judge from the appearances in the post-mortem room there had been an attempt on the part of Nature to prevent this rupture by a process of adhesion between the sac of the aneurysm and the inner wall of the parietal pericardium in contact with it, for in removing the heart from within the pericardium one had to tear apart definite fibrinous adhesions over the aneurysm, obviously dating from a period of several days antecedent to death. Finally, however, the thinning of that part of the heart muscle which was involved in the aneurysm became so great that a little extra exertion of some kind caused spontaneous bursting with rapid death from hæmopericardium.

One is a little apt to forget that the heart may suffer from bruising; and other ill-effects consequent upon accidents without necessarily producing objective signs such as bruits. The patient

may complain after the accident that he has severe precordial pains, and one is, perhaps, too easily led to conclude that these are functional or neurasthenic because of there being few conclusive signs of organic bruising. There may or may not be peculiarities of the pulse rate, especially increased frequency, as in the present instance. The above specimen is a decidedly uncommon one in respect of the degree to which the heart was affected by its local bruising, but it probably exemplifies in an extreme extent that which is not at all uncommon in a less degree.

# ANTE-MORTEM THROMBUS IN LEFT AURICLE PRODUCING A BALL-AND- SOCKET TYPE OF MITRAL STENOSIS WITH PRESYSTOLIC BRUIT.

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By

HERBERT FRENCH.

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THE patient was a young woman, aged 24, who was admitted under the care of Dr. Pitt upon January 3rd for trouble in breathing. As a child she had had scarlet fever; there was no history of acute rheumatism; she had never been very strong. Ten weeks previous to her admission she had a sudden attack of shortness of breath on going to bed, and since that time had suffered from dyspnœa, the latter having become worse during the two or three days previous to her admission. Latterly there had been partial orthopnœa at night. There had been slight hæmoptysis five weeks before she was brought to the hospital. The patient was very wasted as to the body; the legs were slightly œdematous. The pulse was rapid, and irregular both in volume and in rhythm. The heart dulness extended a finger's breadth to the right of the sternum and slightly outside the left nipple line; there was a localised presystolic bruit at the impulse with a loud pulmonary second sound; a few fine râles could be heard at the bases of the lungs. The spleen was not palpable, but the liver came two inches below

the ribs. The urine was normal. A diagnosis of mitral stenosis was made. By January 6th the râles had disappeared and the pulse was more regular. On January 16th the presystolic bruit had disappeared. Towards the end of the month anæmia became more marked than it was on admission, and a blood count showed hæmoglobin 50 per cent. of normal, white corpuscles 3,500 per cub. mm., red corpuscles 3,500,000 per cub. mm. There was irregular slight pyrexia, and it was suggested that the patient might be suffering from malignant endocarditis. On February 21st she vomited twice. On March 4th Dr. Pitt heard a bruit de rapelle, and thought there might be pericarditis. The following day there was a systolic apical bruit, and increased œdema of the left leg set in. A cough developed and rhonchi were heard on the right side behind. The systolic apical bruit was still present on March 3rd, and by this time the abdomen had become distended. On March 13th the patient became very dyspnoëic, and gradually sank and died at 9.15 p.m.

At the post-mortem examination there was only slight œdema, confined to the legs and back. The face was pallid. There were fifteen ounces of serous fluid in the left pleural cavity, twenty ounces of similar fluid in the right pleural cavity, twenty ounces of serous fluid in the abdomen, and eight ounces of bright yellow clear fluid in the pericardium. There was general enlargement and reddening of the lymphatic glands in the thorax and abdomen, especially of those in the superior mediastinum, at the bifurcation of the trachea, in the posterior mediastinum, in the mesentery, and in the gastro-hepatic omentum. None of these glands was less big than a large bean, some were as big as acorns, and all looked bright red as though from some active inflammation. Cultures were taken from the right ventricle and the spleen, and Dr. G. W. Goodhart reported, "Cultivations from these specimens gave a growth of pneumococcus." The lungs exhibited no cardiac induration and no atheroma of the pulmonary arterioles. There was considerable airlessness of the left lower lobe owing to its having been compressed by





*Ante-mortem Thrombus in Left Auricle.*

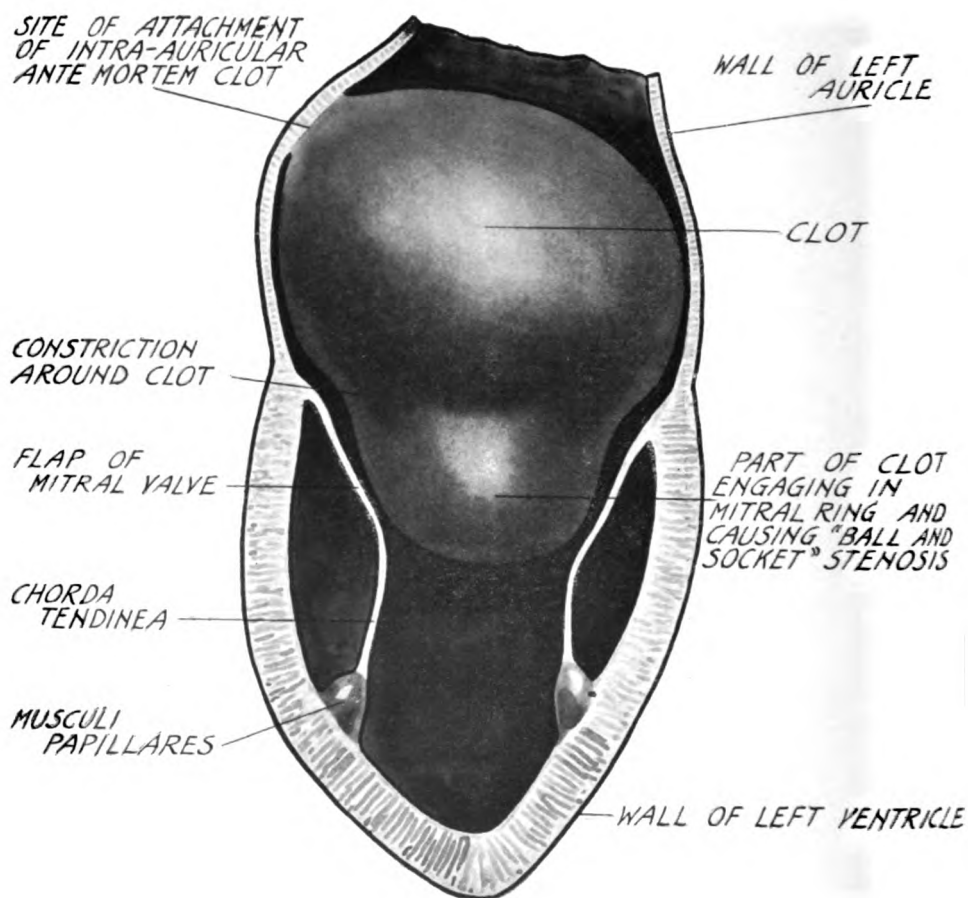


FIG. I.

the much dilated heart, and the lower part of the right lower lobe was partially consolidated by a recent infarct measuring three inches across at the surface. The viscera exhibited the ordinary changes associated with chronic heart failure, the stomach and intestines being deeply congested and livid; the liver typically nutmegged; the gall-bladder œdematous; the spleen weighing 231 grms, firm, dark, and of the cardiac type, without infarct; the kidneys together weighing 264 grms, firm, and of the cardiac type, but not so livid as one would have expected; neither presented any infarct, nor any petechial hæmorrhages of the flea-bitten type beneath the capsule.

The volume of the heart was about half as much again as normal. It was about equally dilated as to all its cavities, auricles and ventricles alike; there was some hypertrophy also, but the dilatation exceeded the hypertrophy. The tricuspid valve in itself was natural, but it was dilated so that the ring admitted four fingers and a thumb to the second joint, indicating that there had been tricuspid regurgitation by dilatation. The pulmonary valves were normal, but the pulmonary artery was so dilated that its girth was just double that of the aorta, namely, just under three inches in circumference, the aorta being only one and a half inches in this case. The mitral valve admitted two fingers and a thumb to the second joint, so that it was neither stenosed nor incompetent, but it had been inflamed formerly as shown by its undoubted opacity, by the fibrosis of the apices of the musculi papillares and by the fusion of many of the chordæ tendineæ together. The valve flaps were not particularly shortened and they were not rigid. There was no evidence of acute endocarditis to be seen on the mitral valve and no fungating endocarditis, but there had almost certainly been former endocarditis. Within the left auricle, however, and firmly attached by a narrow pedicle to the auricular wall there was a mass of clot considerably bigger than an unshelled walnut, its arrangement being depicted to some extent by the accompanying diagram (Fig. I.). There were two portions to the clot: an upper which filled the auricle and a lower which engaged

in the mitral valve; and at the junction between these two parts there was a constriction corresponding with the mitral ring. The clot, on being cut into, was found to be very firm, and mostly pale grayish yellow, but part was also dark red, so that although it was clearly an old clot, it had had additions to its size recently. It was producing a ball-and-socket type of mitral stenosis. How long it had been present it was impossible to say, but it seemed clear that the mass had been responsible for the presystolic bruit heard on admission and for some time after. The disappearance of this bruit was doubtless due to increasing feebleness of the heart muscle, the systolic bruit that developed later being the result of secondary tricuspid regurgitation. The generalised reddening and enlargement of the lymphatic glands was apparently due to a terminal pneumococcal septicæmia which was the direct cause of death.

# ANTE-MORTEM CLOT IN THE RIGHT VENTRICLE ;

PULMONARY STENOSIS OF THE CUP-AND-BALL TYPE  
RESULTING FROM THE GLOBULAR MASS OF FIBRIN  
HAVING BECOME ENTIRELY DETACHED, BUT BEING  
TOO LARGE TO PASS THROUGH THE PULMONARY  
VALVE.

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By

HERBERT FRENCH.

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THE patient was a married woman, aged 44, who was admitted under the care of Sir Cooper Perry on November 18th, 1911, for dyspnœa, swelling of the legs and general weakness, with a previous history of scarlet fever, measles, whooping-cough, and acute rheumatism. The patient had suffered from dyspnœa for five years, and for the last four had been subject to attacks of "asthma." Two years ago she said she had a "stroke" with loss of speech and right-sided hemiplegia. Swelling of the legs began in January, 1911, and had continued since. On admission she was very cyanosed with much œdema of the legs and feet. There was cardiac dulness for an inch outside the left nipple line, but no obvious dulness to the right of the sternum. A soft mid-diastolic bruit could be heard at the impulse, and the second sound was re-duplicated in the pulmonary area, but the heart sounds were weak and muffled, so that it was difficult to interpret them at this time. There was palpable pulsation of the lower border of the liver. Later, a definite pre-systolic bruit developed below the left nipple. Mitral stenosis was diagnosed. The œdema of the legs disappeared, and the

heart became slower and more regular. The heart sounds at the base were very variable, a systolic bruit being audible in the pulmonary area on some occasions and not on others, but there was nothing sufficiently definite to suggest that there was actual pulmonary stenosis. The dyspnoea continued, later, vomiting set in, with recurrence of the heart failure, the patient becoming more cyanosed and generally weaker, and dying on December 11th, 1911.

The body was that of a well-grown woman. The legs were very cedematous up to the knees, but not above them. There was slight cedema of the loins. The lips were so livid that they had the appearance of those of a person who has recently eaten black-heart cherries.

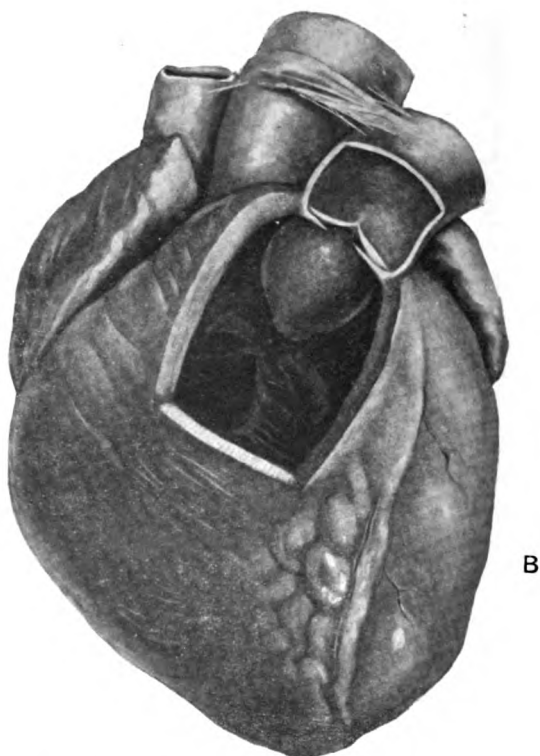
The brain weighed 1,121 grams. There was no obvious lesion in the circle of Willis or in any of its main branches, but on cutting serial sections of the brain substance a large irregular cyst was found in the left hemisphere occupying the whole of the situation of the left lenticular nucleus, the anterior third of the internal capsule, and part of the caudate nucleus on this side: the genu and the posterior two-thirds of the left internal capsule were intact. In length the cyst measured one and a quarter inches; in breadth it was three-quarters of an inch in its maximum diameter, tapering off both anteriorly and posteriorly to nothing. It was not a circular cyst, but rather had the shape of an elongated diamond, and it was clearly the result of the former embolism of which there was a clinical history.

The lungs were typically indurated, red, and very cedematous: in the right lower lobe there were two recent dark crimson hæmorrhagic infarcts; the larger was equal in bulk to about half that of one's closed fist, whilst the smaller was about a third of this size. So large were these infarcts that they caused the right lung to weigh 980 grams, though the left weighed only 514 grams. There was recent blood in the right bronchus, but none in the left.

The pulmonary arterioles were much thickened, standing out prominently from the cut surface of the lung, and when they



*Ante-mortem Clot in the Right Ventricle.*

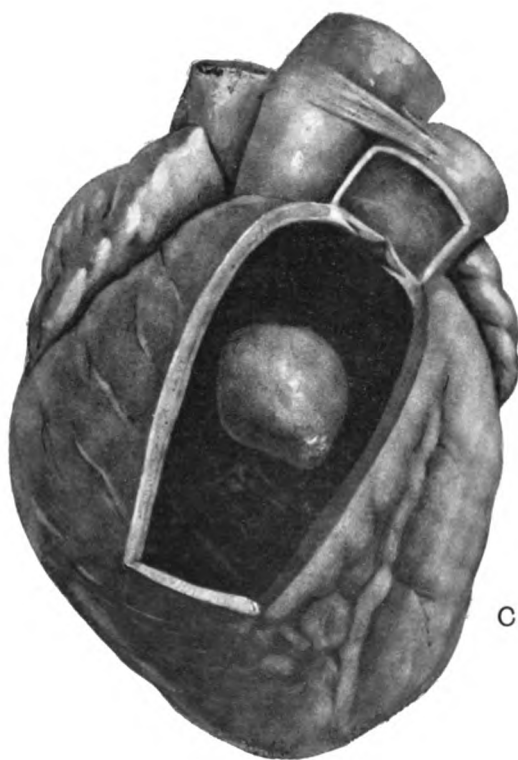


FIGS. A, B, and C, to illustrate pulmonary stenosis of the ball-and-socket type, due to a free globular mass of fibrin in the right ventricle too large to pass through the pulmonary orifice. A. The free mass of fibrin itself, resembling an eyeball, both in shape and size; the part corresponding to what would be the optic nerve is probably the site of a previous peduncular attachment of the blood-clot to the ventricular wall, but the clot itself now lay quite free in the cavity of the right ventricle, and looked as though it had done so for some time. B and C indicate the positions of this free mass within the ventricle in systole and diastole respectively.





*Ante-mortem Clot in the Right Ventricle.*




were slit up they exhibited typical and extensive pulmonary atheroma.

The stomach was empty and purplish crimson from typical cardiac cyanosis; the intestines were similarly livid, but to a less degree; there was no ascites; the liver weighed 1,340 grams, and, though not enlarged, it presented a typical nut-megged appearance; the spleen weighed 148 grams, it was firm and dark: it contained no infarct; there had been former perisplenitis causing it to be adherent to the diaphragm; the kidneys together weighed 270 grams, and each had had multiple infarcts in it on some former occasion, each such infarct being now represented by a deep depression with fibrous scarring; there had been, perhaps, eight such infarcts in each kidney: there was no recent infarction in either; the pelvic organs were natural. The aorta and arteries generally were healthy.

The heart was about twice the normal bulk. The left ventricle was of about the normal size, neither dilated nor hypertrophied. The mitral valve was extremely stenosed and calcareous, allowing the tip of one's little finger just to engage in the orifice, but not permitting even one finger to pass through it. The stenosis was of the button-hole type. There was no terminal endocarditis. The left auricle was enormously hypertrophied and dilated, and its endothelial lining was opaque. The pulmonary artery was irregularly atheromatous in its main branches quite as much as in its arterioles in the lung. The pulmonary valves were normal. Both the right auricle and the right ventricle were very greatly hypertrophied and moderately dilated. The tricuspid valve admitted four fingers and a thumb up to the second joint, so that there had been tricuspid regurgitation; there was no particular thickening or fibrosis of the tricuspid valve itself. The right auricle was even more hypertrophied and dilated than the left auricle, and thickened as to its interior.

The most remarkable point about the heart, however, was the presence within the right ventricle of a spherical, more or less smooth mass of firm old clot (Figs. A, B, and C) about the size and shape of a human eyeball. At one pole of it there was



evidence of its having been attached somewhere to the ventricular wall at a former time, but the detachment had been complete for some while. The mass was just too large to pass through the pulmonary valve, and it is probably owing to the way in which it had alternately engaged in the orifice and then passed back into the ventricle that it had been moulded into its globular shape; this also accounts for the extreme variability of the sounds heard at the base of the heart during life. The mass had produced what one might call a cup-and-ball pulmonary stenosis. For this to be due to what is equivalent to an entirely free foreign body inside the heart is undoubtedly rare. It is not very uncommon to get ball-and-socket stenosis at the tricuspid or mitral valve due to a pedunculated clot attached to the wall of the auricle, as in the case recorded on page 353 of this volume; it is rarer to get similar ball-and-socket stenosis from a polypoid clot extending into the heart from one of the main veins, as in the case recorded on page 379; but to have the clot entirely detached and, as it were, a foreign body within the ventricle is very rare indeed.

In the Liverpool Museum there is a specimen of a heart containing a free globular mass of clot in one auricle causing stenosis at the auriculo-ventricular opening; it is spoken of as "the golf-ball heart." In the ninth edition of Delafield and Prudden's Text-book of Pathology (1912), page 532, figure 329 shows a globular thrombus attached to the wall of the left ventricle by a very narrow pedicle which, if it had become worn through by repeated heart contractions, would have led to the formation of a free globular mass of fibrin in the left ventricle similar to that which was here present in the right ventricle. I cannot, however, find any record of another case precisely like the present one. The free ball of old fibrin could hardly be described better than as being about the size and shape of an ordinary human eyeball with a slight projection corresponding with the optic nerve, presumably the site of the previous attachment of the clot to the ventricular wall. Pulmonary stenosis due to this cause must be very rare even if the specimen is not unique.

# REDUCTION OF BLOOD PRESSURE BY IODIDE OF POTASSIUM.

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By

HERBERT FRENCH.

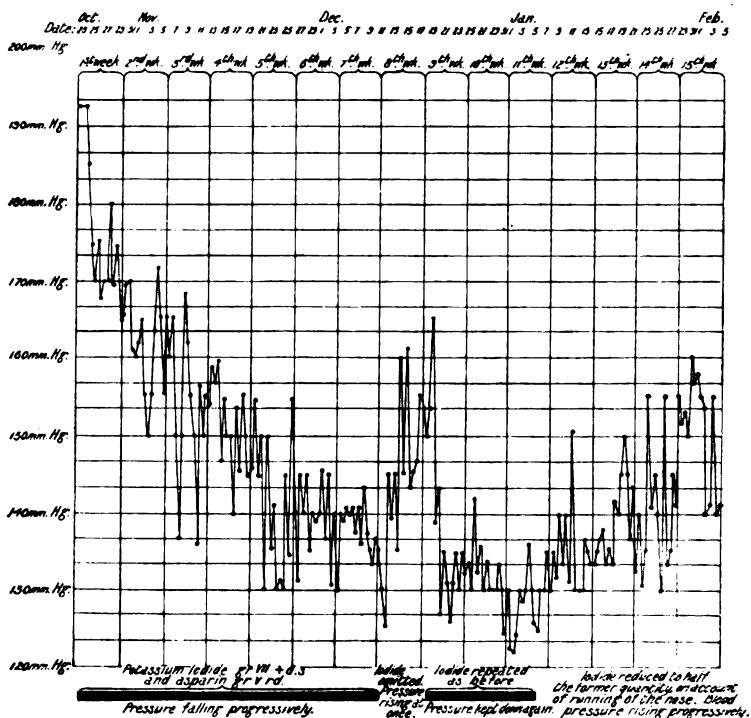
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I do not think it is by any means always wise to take steps towards the reduction of a blood pressure simply because it is high. I have given reasons elsewhere\* why I think that a high blood pressure is, in many cases, particularly of arteriosclerosis, a necessity which needs to be encouraged rather than interfered with. At the same time, there are conditions, such, for instance, as aneurysm of the aorta, in which one is desirous of minimising the blood pressure as much as possible, and it is not every day that one has an opportunity of showing so clearly, as in the accompanying chart, the way in which iodide of potassium can effectually bring the blood pressure down, at any rate in some instances.

The patient was a man, aged 64, suffering from thoracic aneurysm. For this he had been treated for many months by various measures, including complete rest in bed. A time came when he could no longer stay in bed, and although so much of an invalid that he could do no more than be clothed by an attendant and thereafter sit in an easy chair, or be carried

\* *The Clinical Journal*, July 5th, 1911, "The Vaso-motor Factor in the Causation and Treatment of High Blood Pressure"; and the *Lancet*, July 13th, 1912, "High Blood Pressure and the Commoner Affections of Arteries."

from room to room in a carrying chair, he was not confined to bed during the time the following blood pressure readings were taken. The observations extended over fifteen consecutive weeks without any other material alteration than the changes in the iodide given; the blood pressures were read twice daily by means of Messrs. Down Bros.' modification of the Riva-Rocci sphygmomanometer, at 6 a.m. and at 6 p.m.; each reading represents the maximum systolic pressure in the brachial artery.



During the first seven weeks 7 grains of iodide of potassium were given three times a day, together with 5 grains of aspirin, also three times a day, and the blood pressure fell progressively from 190 mm. Hg. to an average of about 140 mm. Hg., on several occasions becoming so low as 130 mm. Hg., and once 125 mm. Hg. The iodide was then omitted, and

almost immediately the blood pressure rose to about 160 mm. Hg. Notwithstanding the fact that the iodide produced a considerable amount of running at the nose, and a tendency to bronchial catarrh, it was deemed advisable to repeat it as before, when the blood pressure was again almost immediately reduced to an average of about 130 mm. Hg.

Three weeks later the running at the nose was so troublesome that it was decided to reduce the dose of the iodide of potassium to a half of that which had been taken hitherto, the average blood pressure rising forthwith to 145 mm. Hg. or more, one reading being as high as 160 mm. Hg.

Diet in this case was the same throughout, and consisted of ordinary foods, though the amount eaten was not large. The bowels tended to be a little costive all the time, and minimum doses of prune paste were given at regular intervals, but otherwise the conditions, except in regard to the administration of iodide of potassium, were approximately the same throughout the whole 15 weeks, so that it would seem that the variations in blood pressure could be attributed directly to the influence of the iodide of potassium: a considerable reduction being brought about by 7 grain doses three times a day; an immediate rise following the omission of the drug; another fall accompanying its re-administration; whilst a diminution of the dose was accompanied by a decided tendency to rise again.



# AN UNUSUAL TYPE OF MELANOTIC GROWTH IN THE LIVER.


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By

HERBERT FRENCH.

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THE patient was a man, aged 56, who died on August 1st, 1905. On October 23rd, 1901, his right eye was excised for a melanotic growth of the choroid coat, presumed to be sarcomatous, though no sections are available of this eye growth itself. The history of the ocular trouble was as follows : The man said that he had always been healthy, though he had had syphilis as a young man; in July, 1901, he felt as though something were "coming over" his right eye, and the sight in it became dim. At first there was also a little pain in this eye, but after the first week or two this disappeared. He attended as an out-patient at Guy's Hospital, and at first was given potassium iodide and perchloride of mercury; as the sight did not improve, but got worse, he was admitted as an in-patient, and for a short time was further treated with mercurial inunctions. On October 29th, however, Mr. Higgins had come to the conclusion that the lesion was a growth; the eye was excised that day, and on cutting into it it was found to contain a large melanotic growth which seemed to be of ordinary choroidal sarcomatous type. The man made an uninterrupted recovery, and went out on December 30th. He was re-admitted on July 22nd, 1905, into John Ward, under the care of Dr. Beddard, giving a history that though he had been well for a year or two after he left the Eye Ward, and





had remained at work, he had been subject during the last two years to repeated attacks of pain in the upper part of the abdomen, these having become more frequent of late. He had become much wasted during the three weeks preceding his admission, and had noticed his abdomen getting bigger, especially in its upper part. He was thin and wasted, with a swollen abdomen, and the liver could be felt very much enlarged, hard, with a firm sharp edge, and without any nodules to be felt in the organ. During life it was thought that the spleen could be felt, but this turned out at the post-mortem examination to have been a greatly enlarged left lobe of the liver. He was obviously very ill, went rapidly down-hill, by July 30th was comatose, and died on August 1st. The post-mortem examination was made the same day five hours after death.

The whole body, including the empty orbit and also the remaining eye, was searched for evidence of a primary melanotic focus, for, at the time of making the examination, the history of the excision of the ocular melanotic growth in 1901 was not known; the patient had been too ill to give a clear account of himself, and, as a matter of fact, he stated that the eye was removed on account of an accident. It was only as the result of subsequent inquiries that the history of the eye trouble was discovered. Not even one pigmented mole could be found on the skin. All the peripheral lymph glands were normal, and there was apparently no melanotic growth in any part of the head or neck. There were no secondary deposits in the lungs or pleuræ.

The heart weighed 400 grams, and exhibited acute terminal endocarditis of the aortic valves, but no other abnormality.

The liver was enormous, weighing just over 14 lbs. The appearances were very remarkable, and were entirely different from that which one generally associates with secondary melanotic growth. In addition to a number of ordinary nodules of pale growth externally containing streaks of black pigment and surrounded by melanotic tissue, there was also elsewhere a widespread continuous infiltration of almost the whole liver tissue

with coal-black material, without any distinct breaking up of this into separate deposits or nodules. The enlargement of the liver was fairly uniform except that the left lobe, mistaken clinically for spleen, was more hypertrophied than the right, and on cutting into the liver it was hard, and internally for the most part as black as a piece of coal. In the left lobe this blackness was uniform and universal, the appearances being what one might describe best as those that would be presented by a liver that has been dyed through and through with black ink. Dr. Hale White suggested at the time the post-mortem examination was made that the condition might be one of primary melanotic carcinoma of the liver; in view of the history of the eye trouble, ascertained after Dr. Hale White made this suggestion, one would be inclined to regard the melanotic growth in the liver as secondary in some way to that in the eye, although no connecting deposit of new growth could be discovered. It is particularly noteworthy, therefore, that when the sections of the liver growth were examined microscopically by Mr. Targett, he reported that they showed "melanotic *carcinoma* consisting of rather large polyhedral cells arranged in the form of narrow elongated alveoli with pigment distributed unevenly throughout the section." It is unfortunate that there is no section of the original growth in the eye to show whether this was sarcomatous or carcinomatous.

The other viscera were examined with particular care; the only other secondary deposits found were in one or two black minute glands in the front part of the diaphragm and also a few other similar glands in the head of the pancreas; each of these was no larger than a pea, but so black that they attracted notice at once, and they were found microscopically to contain secondary melanotic growth similar to that in the liver.



# A CASE OF ADRENAL HYPERNEPHROMA IN A YOUNG GIRL

WITH DEVELOPMENT OF PUBIC HAIR AND HYPER-  
TROPHY OF THE CLITORIS IN INFANCY.

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By

HERBERT FRENCH.

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THE clinical and pathological characters of renal and adrenal hypernephromata have been discussed by various authors of recent years, one of the newest and fullest papers upon the subject being by Dr. Ernest E. Glynn, entitled "The Adrenal Cortex, its Rests and Tumours; its relation to other Ductless Glands, and especially to Sex," in the *Quarterly Journal of Medicine* for January, 1912, vol. v., pp. 157-192. He gives abundant references to the literature, and there is no need to repeat them here. The total number of recorded cases of the malady is small, however, Dr. Bulloch and Dr. Sequeira having collected 12 up to 1905 and Dr. Glynn having added 5 more, including brief notes of the present one. It seems right, therefore, to add to the number whenever opportunity arises, and it is with this object that the following details are given:—

The patient, when admitted to Guy's Hospital on August 18th, 1910, under the writer's charge was  $6\frac{1}{2}$  years old. She came in on account of acute pain in the left side of the thorax due apparently to local inflammation over her suprarenal tumour. She gave a history of having been a very healthy child, except that since one year old she had been subject to periodic attacks of vomiting about three times a year, these bouts becoming

rather more frequent during the last two years, and sometimes lasting continuously for three days at a time. Pubic hair was noticed when the child was eight months old; for two years it increased in amount steadily, but since then it had remained almost stationary. There was no abnormal hair in the armpits nor on the face. During the six months before admission she had complained occasionally of pain in the "stomach," more especially on the left side. On the night of August 14th, 1910, she was suddenly seized with great pain in the left side of the abdomen below the left costal margin, accompanied by retching and vomiting. The pains were relieved during the next day or two by medicines and hot external applications, but as they did not cease entirely, and the child was clearly ill, she was brought to the hospital and at once admitted.

The accompanying photographs show a healthy-looking child, bright and intelligent, without any tendency to increase in the size of the breasts, without undue fatness, but with the remarkable premature development of the pubic hair which is shown in Fig. I., and the equally remarkable hypertrophy of the clitoris shown in Fig. II., which was taken after the pubic hair had been shaved off. A relatively large tumour was felt with ease in the left side of the abdomen, descending from beneath the left hypochondrium and also extending into the left flank. The mass moved up and down with respiration; it presented a rounded lower pole which extended for about the breadth of one's hand below the ribs, but one could not get between the mass and the costal margin to feel its upper pole. It felt smooth and free from either nodules or notches. It was very tender on the least pressure. It could be palpated readily in the loin bi-manually, and could even be seen to fill out the ilio-costal space a little, as a renal tumour would. It extended nearly as far as the middle line in front just above the umbilicus, but did not cross to the right. There was resonance over the front of the tumour, but it was dull to percussion both on its left side and posteriorly; this posterior dullness was continuous with a marked impairment of note at the

*A Case of Adrenal Hypernephroma in a Young Girl.*

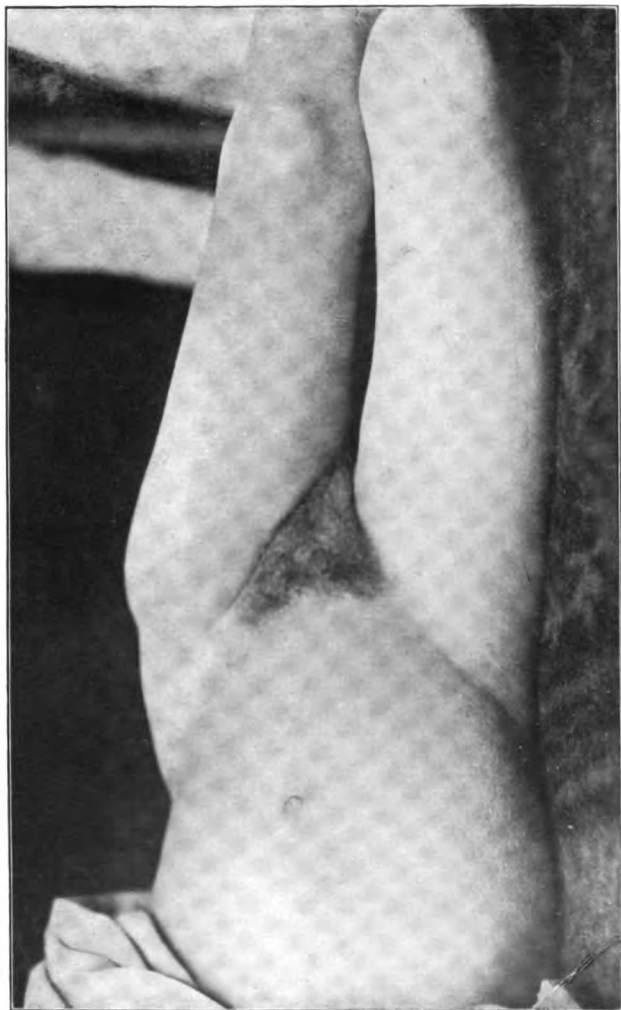


FIG. I.—This shows the pubic hair which had been present since the girl was eight months old.



*A Case of Adrenal Hypernephroma in a Young Girl.*




FIG. II.—The pubic hair has been shaved. The figure shows the hypertrophy of the clitoris, the absence of hypertrophy of the breasts, and the absence of axillary hair. The general development and the facial appearance correspond with the patient's age.





base of the left lung behind, normal pulmonary resonance not being reached until the level of the seventh rib, and there was corresponding diminution of vesicular murmur and of voice sounds, suggesting that the mass was pushing the diaphragm upwards and compressing the lower lobe of the left lung. Examination with the x-rays confirmed this; the left half of the diaphragm moved very little, and in addition to the mass seen below there was an ill-defined shadow above it also, suggesting pleurisy with a small amount of effusion. No pleuritic rub could be heard, however. The urine had a specific gravity of 1030; it was high-coloured and scanty, but contained neither albumen, blood, nor sugar.

Cystoscopic examination showed that urine was descending both ureters normally, so that hydronephrosis could be excluded. The results of blood examination were negative, the red corpuscles and hæmoglobin being only a little below the normal, the leucocytes 13,000 per cub. mm., and the differential leucocyte count natural. A diagnosis of adrenal hypernephroma was made. At first there was slight pyrexia and a rapid pulse (Fig. III.) due, like the pain in the left side, to pleurisy over the secondary deposits in the lungs no doubt, though these were not diagnosed at the time. Consultations were held as to the best procedure to follow, and it was finally decided to perform laparotomy with a view to removing the tumour if possible. Mr. R. P. Rowlands operated on September 1st. He found the mass to be an adrenal tumour which almost completely surrounded the left kidney, making it impossible to remove the one without the other. He took out the whole mass which, including the kidney embedded in it, was nearly the size of one's two closed fists. The adrenal tumour itself was of a curious yellow-ochre colour, very vascular, and so soft that it broke down with great ease under digital pressure. The growth, though it was creeping down all over the capsule of the kidney, did not invade the latter at all. The kidney itself looked perfectly normal. The specimen was sent to the Gordon Museum for preservation.





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*A Case of Adrenal Hypernephroma in a Young Girl.*

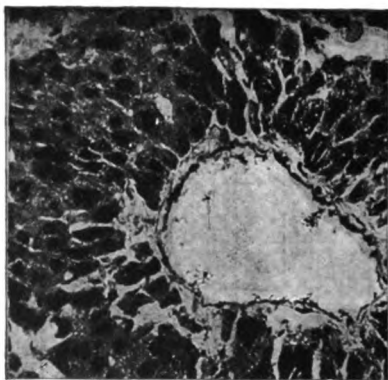


FIG. IV.—A small portion of the Adrenal Hypernephroma seen under the high power, showing the perivascular arrangement of the columns of polyhedral cells of the new growth.

metastases in the lung. At the post-mortem examination both lungs were found to contain numerous secondary deposits, varying in size from quite small dots to globular masses half an inch in diameter, all of a yellow-ochre colour similar to that of the adrenal growth itself, together with crimson hæmorrhages into most of them. There was pleurisy over the left lower lobe, but no measurable quantity of free fluid. In the right lung Dr. C. H. Rippmann, who performed the autopsy, demonstrated that the growth was present in the pulmonary arterioles, whence it was invading the surrounding lung. There were no other secondary deposits. It was specially noted that, notwithstanding the pubic hair and the hypertrophied external genitals, the uterus and the ovaries were perfectly normal, which is the rule in these cases.

Microscopical sections both of the primary neoplasm and of the secondary deposits in the lungs showed a very vascular necrosing growth consisting of large polyhedral cells, separated into solid groups by a fine connective tissue stroma. The appearances were like carcinoma at first sight, but here and there the growth was arranged perivascularly in so decided a way that one would call it a perithelioma of mesoblastic origin rather than a carcinoma. This perivascular arrangement of the neoplasm in the present instance is illustrated in Fig. II from Plate 8 of vol. v. of the Quarterly Journal of Medicine (*loc. cit. supra*), kindly lent by the Editors of that Journal (Fig. IV.).

It is a remarkable feature of these cases that the original tumour may apparently remain localised for years and yet lead to extensive secondary deposits in the end. The present patient had pubic hair when she was eight months old. It is difficult to suppose that the causal hypernephroma of the adrenal gland was not already present then, yet apparently it was not until the child was between six and seven years old that malignant metastases developed in the lungs.



# CALCAREOUS CONCRETIONS EXPECTORATED IN THE SPUTUM IN A CASE OF PHTHISIS IN A YOUNG MAN.

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By

HERBERT FRENCH.

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CALCIFICATION in the scars of obsolete healed phthisis is a familiar finding at the apices of the lungs in the post-mortem room; similar calcification in the tissues round the bronchi at the roots of the lungs has been demonstrated to be common by Dr. Jordan\* and others using the X-rays. It is perhaps surprising, therefore, that persons who are the subject of phthisis do not more frequently show evidence of having had tuberculous mischief in the lungs before by expectorating calcareous concretions or pellets derived from foci which, having previously healed, become eroded again when the tuberculous process recurs. One does not meet with the symptom very often, though probably only a very small number of those cases in which it does occur find their way into the literature. A case in point is recorded under the heading of "Pulmonary Concretion expelled during a Fit of Coughing," in Epitome No. 354, British Medical Journal, part II., December 16th, 1911, page 89. This abstract is verbatim as follows:—

"Brandeis (Gaz. hebdom, April 23, 1911) relates this unusual occurrence as having happened to a doctor aged 43. Both the

\* Peribronchial Phthisis. The British Medical Journal, August 31st, 1912.



patient's parents had died of tuberculosis, and he himself had suffered from tubercle of the right apex, but this had become arrested. There was some chronic bronchitis, with bronchiectasis on the right side, but the left lung was normal. Expectoration was abundant, thick, and inodorous, and an examination revealed neither tubercle bacilli, Charcot-Leyden crystals, nor elastic fibres. There were, however, many gram-staining organisms. One day during a fit of coughing the patient felt the presence of something unusual in his pharynx, and spat out a large mass, dark green in colour, swollen at one extremity and thinned out at the other. At the thinner end there was buried a hard mass, dry and brittle in consistence, which, on micro-chemical examination was found to consist of phosphates, fibrin, and hæmoglobin. There were no other organised elements. A second rather smaller mass followed. The author expressed his difficulty in determining the nature and origin of this concretion. It may be, he suggests, a portion of a small bronchus and necrosed pulmonary parenchyma in the immediate vicinity; or perhaps a pseudo-membranous cast of a bronchus which had undergone a sort of mummification by prolonged retention in the bronchus, with secondary infiltration by mineral salts. Again, the mass may be an old tuberculous process which has undergone calcareous transformation. The author concludes by saying that he considers the case worth mentioning as adding to the limited number of cases of pulmonary lithiasis which have been reported."

A not very dissimilar case occurred to a patient who was sent up by Dr. J. M. Tudge, of Cricklewood. The patient was a student 21 years of age, an only child whose mother died of carcinoma of the rectum, whose father was alive and well, but whose father's brother died of phthisis. There was no history of tubercle on the mother's side. He himself was a well-built youth, who had taken part in rowing and other exercises, but who had also worked exceedingly hard and had taken scholarships at school and at the University. He had for many years been subject to nasal catarrh and coryza in the spring and summer, this being apparently ordinary hay fever. In March,

1910, he took cold and developed a cough lasting a few weeks; there was no expectoration at this time, the chest was examined very carefully by a physician who could find no abnormal physical signs, and a provisional diagnosis of bronchitis was made. For the sake of safety he went away to the seaside for a while and appeared to get perfectly well; but after working very hard for an examination in March, 1911, he remained below par during the early summer, and was still not feeling himself when he came back from his summer holiday. He had not lost a great deal of weight, but it was found that he had an evening temperature of about 100° F. taken in the mouth; he had several times been waked up by fits of coughing during the night, and there was a small amount of muco-purulent sputum. Tubercle bacilli were found in the latter, and in September, 1911, there was definite impairment of note on the right side, above the clavicle, on the clavicle, and in the first, second, and third spaces, this impairment getting progressively less from above downwards, whilst on auscultation there were fine crepitations over the whole of the right upper lobe in front without bronchial breathing or pectoriloquy, but with bronchophony and deficiency of breath sounds. Posteriorly there were definite and similar crepitations over the right supraspinous fossa and fine crepitations over the uppermost inch of the right lower lobe behind. On the left side no definite abnormal signs could be detected. He was treated first at home and later at various seaside resorts; he gained weight, ceased to have evening pyrexia, lost the abnormal crepitations, though the impairment of note over the right upper lobe still persisted. Tubercle bacilli ceased to be discoverable in the sputum, and at the end of a year he was able to return to his work again, though he still adopted special precautions for keeping well.

The interest in the case lies in the fact that on October 3rd, 1911, after a little cough he felt something come up into his mouth which seemed different from the ordinary sputum, and he had the curiosity to keep it. It was reddish-green in colour, but otherwise exactly like an ordinary nummular mass of

phthisical expectoration. On feeling it between the fingers, however, something hard could be felt within it, and on picking the hard substance out from the interior of the mass it was found to be an irregular calcareous particle of which No. 1 in the accompanying diagram (Fig. I.) is a life-size representation. On October 20th after another similar bout of coughing he again felt that the pellet of sputum in his mouth contained something hard. This broke in two in extracting it from the mucous pellet in which it was embedded, and it is represented by No. 2 in the accompanying diagram. A little later a third calcareous concretion was expectorated in a similar way, but this proved so brittle that by the time it was extracted it was too comminuted to keep. There were no more calcareous particles in the sputum until the 22nd April, 1912, when, although the patient had increased 21 lbs. in weight and was walking as much as 12 miles a day, apparently without any ill-effects, he coughed up a little sputum containing the calcareous particle marked 3.

Dr. J. H. Ryffel kindly analysed them, and reports that "they consist of calcium phosphate mainly, with some carbonate and a little organic matter."

One cannot but suppose that although this patient's illness appeared to begin with the attack of bronchitis in March, 1910, he had really suffered from tuberculous mischief in the lung, perhaps years previously; that this previous attack had resulted in temporary cure with calcification of the caseous material in the resulting scar; and that when the phthisis recurred the original healed focus became slowly eroded by the fresh mischief, thus liberating the calcareous particles which were found in the sputum.

*Calcareous Concretions Expecterated in the Sputum.*



FIG. I.

CALCAREOUS CONCRETIONS FROM PHTHISICAL SPUTUM.

ACTUAL SIZE.

1. Expecterated on October 3rd, 1911. 2. Expecterated on October 20th, 1911. 3. Expecterated on April 22nd, 1912.



# CARCINOMA OF THE LEFT KIDNEY

WITH CONTINUOUS CLOT EXTENDING FROM THE KIDNEY ALONG THE LEFT RENAL VEIN AND UP THE INFERIOR VENA CAVA INTO THE RIGHT AURICLE, PRODUCING AN INTER-CARDIAC POLYPOID MASS WHICH LED TO TRICUSPID STENOSIS OF THE BALL-AND-SOCKET TYPE, THE CLOT BEING INFILTRATED WITH CARCINOMA OF THE SAME TYPE AS THAT IN THE KIDNEY ITSELF.

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By

HERBERT FRENCH.

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## CLINICAL NOTES OF THE CASE.

THE patient, a man aged 45, was admitted to Stephen Ward, Guy's Hospital, on January 21st, 1911, and died on February 26th, 1911. He came in for indefinite but severe abdominal pains, and also complained of cough. He was a married man who had four healthy children and who gave no family history of phthisis or malignant disease. Eighteen years previously he had lain in the London Hospital suffering from a fracture of the base of his skull, and had made a complete recovery. He stated that he had had influenza followed by pleurisy  $3\frac{1}{2}$  years ago, but that otherwise he had always been strong and well. His own account of his present illness was that it had started suddenly at the end of September, 1910, fifteen weeks before his admission to the hospital. He came home from his work one day apparently in perfect health, had his dinner as usual, and immediately afterwards was seized with acute abdominal pain, accompanied by profuse diarrhoea and sweating, the attack lasting about two hours. He could not say

in what part of the abdomen the pain had been, but he seemed to think that it had been all over and not more marked on one side than the other. Since that primary attack he had had alternating diarrhoea and constipation with intermittent abdominal pains. He described a typical attack as follows: He would wake in the morning and find himself coughing without being able to bring up any sputum, and at the same time he would be suffering from pain in the region of the umbilicus, this pain becoming steadily worse as the day wore on, though he would be able to eat both his breakfast and his mid-day meal. He found that half a glass of hot rum during the morning would give some relief, but by the evening the pain would be so bad that he would have to go to bed between hot blankets and with hot applications to his abdomen, by means of which he obtained some relief. There would be no vomiting, but the sensation of extreme abdominal distension would become more and more marked, and he would then take some laxative medicine from which an attack of diarrhoea generally resulted, and at the same time the pain would be relieved. As the diarrhoea produced by the medicine subsided, constipation would ensue and persist until the next attack, which would take place in a few days. He had never noticed any blood in his motions nor anything the matter with his water. The abdominal pain was accompanied also by pain between his shoulders about the level of the sixth dorsal vertebra or rather lower, and extending thence all down his back to the lumbar region. Paroxysms of coughing were also very troublesome; they occurred chiefly at night and in the early morning and they were sometimes so violent as to cause him to retch repeatedly, though he never brought up any obvious sputum and did not vomit. Of late he had frequently found himself sweating profusely when he woke in the night, and he had also begun to suffer from headaches to which he had not been subject previously. He used to be a heavy smoker, but had not enjoyed his pipe of late, and had, therefore, given up smoking to a considerable degree. He denied having had syphilis; he had been a moderate drinker, but had never taken alcohol to excess.

On admission he was obviously unwell, looking thin, pale, and drawn about the face, and he stated that he had lost a great deal of weight lately. His teeth were in a very bad state; this, however, is almost invariably the case with hospital patients of his class, so that little could be concluded from them. There was considerable tenderness on palpating the abdomen, and one thought that this tenderness was most marked on palpation over any part of the large intestine, especially over the cæcum and ascending colon, but no lump or other definite abnormality could be made out at this time. The urine was slightly clouded, of specific gravity 1024, and acid reaction; it contained a trace of albumen, but microscopically no blood, no renal tube casts, and no obvious pus, nor were there any crystals. The urine contained no sugar.

The lungs presented no very obviously abnormal signs, although it was thought that the resonance over the first and second right spaces in front was less good than the corresponding resonance on the left side. There was no bronchial breathing, bronchophony, or pectoriloquy, nor could any râles or rhonchi be heard. There was a very little mucous sputum once or twice, and this was examined carefully, but no tubercle bacilli could be found.

The cardiac impulse was felt faintly in the fifth left intercostal space in the mid clavicular line, and the cardiac impairment of resonance extended upwards to the upper border of the third left rib, to the left as far as the left nipple line, and to the right as far as the left border of the sternum, there being no extension across to the right of the sternum. The heart sounds were peculiarly indistinct over the whole cardiac area, but throughout the illness in spite of repeated examinations no cardiac bruit could be detected. As time went on attention was readily attracted by the remarkable rapidity of the heart's action and frequently by its irregularity.

The following represent his temperature, pulse, and respiration charts:—





As regards the nervous system, the knee-jerks seemed natural. There was no optic neuritis or other retinal change. The sphincters were unaffected, and the only abnormality noted was that whereas the right plantar reflex was briskly flexor that upon the left side was decidedly extensor, this being confirmed upon many successive occasions. There was a marked degree of anaemia, the red corpuscles amounting to 2,767,000 per cubic millimeter, the white corpuscles to 12,700 per cubic millimeter, and the haemoglobin to 55 per cent. of normal by Haldane's method. The differential leucocyte count gave polymorphonuclear cells 78 per cent., small lymphocytes 13 per cent., hyaline corpuscles 7 per cent., eosinophile corpuscles 2 per cent. The red corpuscles in the films did not look unnatural as regards size and shape.

The correct diagnosis during life was by no means obvious, and was not made previous to the post-mortem examination. The first impression in the case was that there was an inflammatory lesion of the colon, and the different forms of colitis most discussed were malignant, tuberculous, and typhoidal. Of these, owing to the impaired note at the right apex, the severe paroxysms of cough that the patient suffered from, and his night sweats, wasting, and pyrexia, the tuberculous met with some favour at first; but no tubercle bacilli could be found in the sputum: von Pirquet's skin reaction was negative; and examination of the thorax with the x-rays showed no obvious degree of shadowing of the apices notwithstanding the slight but definite impairment of note on percussion there. The next suspicion was that the mischief was typhoidal, and a specimen of blood was tested in the Bacteriological Laboratory for Widal's reaction, and curiously enough it was found positive in dilutions in both 1-50 and 1-200. For a time, therefore, typhoid fever was thought to be the diagnosis, though the long history and the relatively fast pulse rate, together with the absence of spots and the rather abnormal character of the temperature chart always rendered this diagnosis doubtful. A third diagnosis which found favour for some time was that of malig-

nant endocarditis; the remarkable rapidity of the heart's action, the peculiarity of the sounds, their irregularity and the degree to which they were muffled although no actual bruit could be heard, seemed to support this diagnosis, and it was thought that possibly there had been an embolism of one of the mesenteric arteries to produce the abdominal pains with alternating diarrhoea and constipation. Blood cultures yielded a mixed growth of *streptococcus pyogenes longus* and *staphylococcus aureus*, and this seemed to confirm the diagnosis.

By February 9th the patient was sitting up in bed and looking better; he did not look so anæmic, had no pain, and was sleeping well, though he was still troubled a good deal with his hacking cough without much sputum.

By February 15th the liver had swollen, and although it did not seem abnormal in shape it could now be felt one and a half inches below the costal margin; and at the same time in the left flank between the antero-superior iliac spine and the left costal margin, a distinct resistance was met with on bimanual palpation, and a not very definite tumour could be felt there, with considerable tenderness on deep palpation. Five days later, on February 20th, this swelling in the left flank was more definite, and as it did not seem to fill out the loin, but rather appeared to come down from beneath the costal margin, between which and the tumour it was not possible to get the hand, it was thought to be an enlarged spleen in which there had been an infarct the result of embolism from the supposed malignant endocarditis; though an alternative suggestion favoured by some was that this was a carcinoma of the splenic flexure of the colon to which the alternating attacks of diarrhoea and constipation might be attributed. There was no complete dullness behind the tumour, and it appeared to be dull in front, so that though kidney was discussed this seemed to be definitely put out of court and the correct diagnosis was thereby missed.

On February 22nd the patient vomited once for the first time. On the 23rd he vomited twice, and on the 24th February he was obviously much weaker. He vomited several times during the

night between February 24th and 25th, and was quite unable to keep down either food or medicine. Shortly after this he became too weak to vomit; was cold, and very feeble with a sub-normal temperature; and he died at 5 p.m. on Sunday, February 26th, having been conscious to within half an hour of his death.

#### POST-MORTEM EXAMINATION.

At the post-mortem examination it was noted particularly that there was no œdema anywhere.

The brain weighed 1,400 grams and seemed perfectly natural; the calvarium was a good deal thicker generally than the average, but otherwise it seemed healthy and no trace of the former fracture of the base of the skull, for which the patient had been treated in the London Hospital 18 years previously, could be detected.

The lungs exhibited numerous fibrous adhesions over the right upper lobe, both in front and behind, and these were doubtless responsible for the impairment of note without radiographic mottling during life. There was no phthisis nor any consolidation of either lung, and it is remarkable that in spite of the extent of the clot in the inferior vena cava and right auricle there was no sign of any embolism or infarction of the lungs. There were no secondary deposits in either the lungs or pleuræ, no recent pleurisy, and the lymphatic glands throughout the body were natural. Apparently there were no metastatic deposits anywhere, only the growth in the kidney and its continuation directly into the renal vein, the inferior vena cava, and the right auricle. The pericardium was natural.

The heart was not weighed because, together with the inferior vena cava, the left renal vein, and the left kidney, it was kept entire for museum purposes. It was moderately dilated as to its right side, natural as to its left, and the mitral and aortic valves were healthy. There was no infective endocarditis of any of the valves. The coronary arteries were healthy and the aorta quite

free from atheroma. In the right side of the heart, however, there was a remarkable polypoid mass comparable to that recorded in some cases by Dr. Owen Richards, and it may best be described by starting with the left kidney. The latter in the fresh state was about twice its normal bulk, and when the capsule was peeled the organ was found to contain a soft umbilicated new growth replacing approximately one-quarter of the renal substance, leaving more or less renal tissue at both the upper and the lower poles, but in the intervening part entirely replacing the kidney substance from renal pelvis to cortex. Superficially the growth protruded about a quarter of an inch above the surrounding renal substance; and it was of a pale yellowish-brown colour, but with an indefinite line of demarcation between itself and remaining kidney substance. The neoplasm had directly infiltrated the kidney venules and by direct extension into the left renal vein had produced thrombosis in the latter, the very firmly adherent clot being in its turn infiltrated by new growth. This process of direct extension of clot and infiltration of the resultant thrombus by new growth extended from the renal vein into the inferior vena cava, as is shown in the illustrations (Figs. I. and II.) taken from the specimen. Below the level of the left renal vein the inferior vena cava was quite patent, but from the left renal vein up into the right auricle there was a continuous solid column of firm clot, attached to the vessel wall in part of the circumference of the latter, but not all round its lumen. This clot extended directly into the right auricle filling it out with a thick elongated moulded mass of clot which in size and shape it is very difficult to describe, though if I may be pardoned for making what may seem to be a rather vulgar comparison it bears a rather remarkable resemblance to an ordinary full-grown penis, and the part that corresponds with the glans was projecting into the tricuspid orifice and engaging in it in such a way as to produce a marked degree of stenosis of the ball-and-socket type. The illustration only partly represents the remarkable characters of the clot and polypus as seen in the post-mortem room.

*Carcinoma of the Left Kidney.*

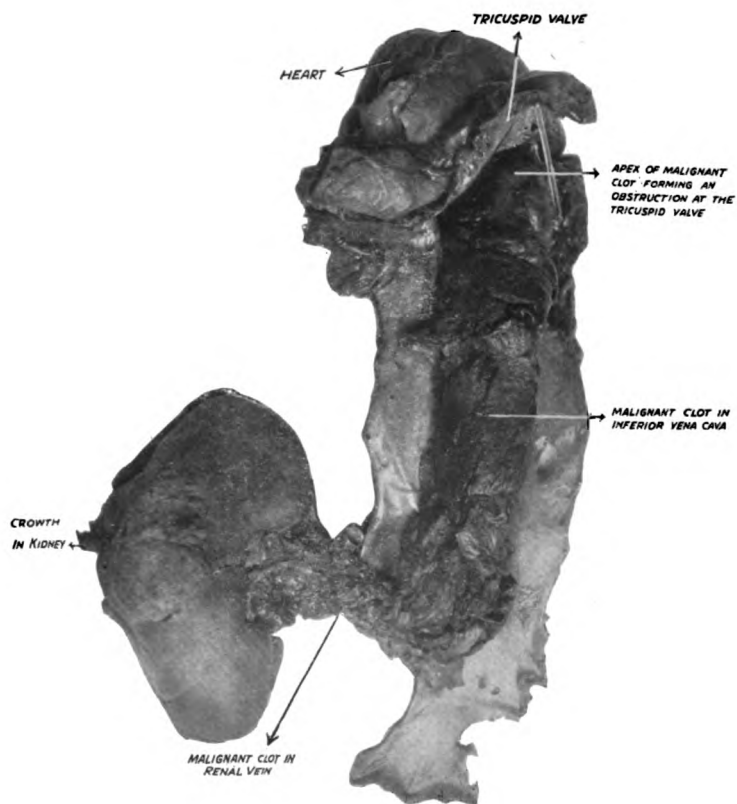


FIG. I.—From a photograph of the specimen.



*Carcinoma of the Left Kidney.*

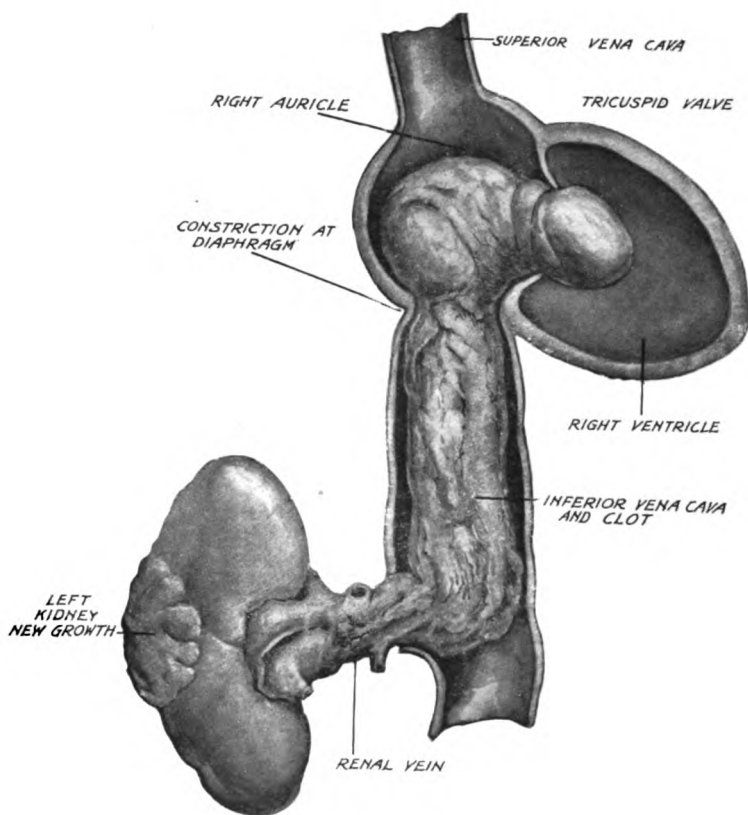


FIG. II.—From a drawing which shows the relationships more clearly than the photograph does.





It is particularly noteworthy that there was no œdema of the legs in this case, and also that there was no pulmonary embolism. The enlargement of the liver noted as coming on during life was clearly due to obstruction of the hepatic veins by extension into them of clot from the thrombus in the inferior vena cava, and this was demonstrated in the post-mortem room, though it was impossible to keep the liver attached in the museum specimen. The spleen weighed 248 grams, and it came below the costal margin sufficiently to be palpable, and the cause of this enlargement was presumably obstruction to the flow of blood from the spleen owing to the clotting of the hepatic veins which had led in its turn to backward pressure in the portal circulation. The mass felt during life was demonstrated to be made up of both spleen and left kidney, and it was the spleen rather than the kidney that constituted the mass that had been felt.

The stomach and intestines exhibited no abnormality except passive congestion, and it seems clear that the abdominal symptoms that the patient had suffered from during life were the result of the inferior vena cava thrombosis and not of a gross lesion of the intestines themselves. It is surprising, perhaps, that there had not been some more definite urinary symptoms in this case, but it is likely that most of the secretion was coming from the normal right kidney, the left, owing to the complete obstruction of the left renal vein, probably supplying very little of the total amount of urine passed. There had, however, been nothing during life to indicate the possible value of cystoscopy in this connection.

Looking back at this case it seems obvious that if a precisely similar patient should be met with again the diagnosis would be as little likely to be made correctly as in the present instance unless a definitely renal tumour could be palpated or unless the inferior vena cava, besides containing a large column of clot, became entirely obstructed by this clot so as to lead to œdema of the legs and back, instead of having a pervious channel left between the thrombus and the vessel wall.

The streptococci and staphylococci found in the blood during life also made the diagnosis difficult; doubtless they were due to some intercurrent infection. It is worthy of note that cultivations taken from the spleen post-mortem gave a pure growth of staphylococcus aureus, so that the staphylococci found in the blood during life were presumably not due to contamination from the skin in obtaining the blood for culture, but were really in the blood stream.

The microscopical characters of the growth in the kidney and in the inferior vena cava showed that the tumour was a spheroidal-celled carcinoma. Fig. I. is a photograph of the specimen, and Fig. II. is a diagram to indicate schematically the conditions as they presented themselves when the organs were in the fresh state.

The following are the reports upon the microscopical sections of the renal tumour and of the clot in the inferior vena cava respectively:—

*Section of the growth in the left kidney.*—Only here and there in this section can any of the renal glomeruli and tubules be detected; the greater part of the section consists of a bold net-work of fibrous tissue in which remains of renal glomeruli can still be seen, very much elongated as though they had been drawn out by tension in one direction by the stretching produced by the new growth. In the meshes of the fibrous net-work there is an exceedingly cellular new growth, the cells of which vary considerably in size and shape though they are all of the polyhedral variety. The growth is a polyhedral-celled carcinoma.

*Section of the growth in the inferior vena caval clot.*—This section of clot in the inferior vena cava is extensively infiltrated with polyhedral-celled carcinoma precisely similar to the primary growth in the kidney.

# A SIMPLE METHOD OF LETTING BLOOD BY MEANS OF A SHORT, WIDE, HOLLOW NEEDLE,

THE NEED FOR SUBSEQUENT DRESSING OF AN  
INCISION BEING THEREBY AVOIDED.

ALSO THE VALUE OF THE SPHYGMOMANOMETER  
ARMLET AS A TOURNIQUET FOR VENESECTION.


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By

HERBERT FRENCH.

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THE heading of this paper is, by itself, almost sufficient to indicate all the main points it contains. Venesection as a therapeutic measure went almost completely out of fashion after it was used to riotous excess in the earlier part of the nineteenth century. It is, however, coming into vogue again in certain maladies, particularly in cases of heart failure with lividity, such as may result from valvular heart lesions such as mitral stenosis or from other causes of over-distension of the right side of the heart, such as chronic bronchitis and emphysema; in certain cases of acute lobar pneumonia in plethoric individuals; in the treatment of uræmia or puerperal eclampsia; threatening diabetic coma; and it is also of considerable value as a periodic blood-letting in the treatment of certain cases of arterio-sclerosis or granular kidney with high blood pressure. When a patient has to be confined to bed in any case it does not matter so very much whether, as the result of venesection, he has to have the



site of the operation dressed or not, but if venesection has to be used in the treatment of high blood pressure in individuals who do not wish to have their daily work interfered with, then it is a very great advantage to have a means of letting blood without the need for any incision of the skin. By the use of a hollow needle it is a simple matter to allow as much blood to escape from a vein as one wishes without having more injury to the skin than what is left by the needle prick. The ordinary needles used in obtaining blood for purposes of blood culture, however, are generally of a bore so small that the blood runs through them too slowly to prevent clotting in the needle before any considerable number of ounces have been withdrawn; and the length of the ordinary blood culture needles is another objection to their being used for simple venesection. Messrs. Down Bros. have made for me some much shorter needles of wider bore; in length from two to four centimeters and in diameter two millimeters (Fig. I.). There is no reason why, for patients with particularly large veins, a needle of even bigger bore should not be employed.

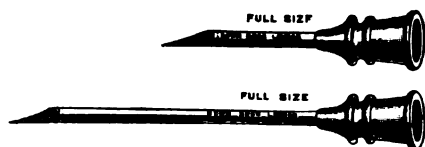


FIG. I.

Having applied the tourniquet to the upper arm so as to distend the veins without obstructing the arteries, and having cleaned the skin over the site of venesection with a little ether, this short stout hollow needle is simply plunged into one of the big veins, the point being directed downwards towards the hand in order that the blood flowing up the veins may enter directly into its open end. This is the reverse of the usual direction in which the needle is used in ordinary blood culture work, but it is a material point if more than a small quantity of blood is to be removed. Dark venous blood spurts through the needle

and continues to run with considerable rapidity. It is collected in a measure glass in the ordinary way, and as soon as it is desired to stop the flow the tourniquet is removed, the needle taken out, the arm held vertical for a moment or two, a little dry boric acid powder is sprinkled over the site of venesection, and no further dressing is required. The procedure can be carried out perfectly well in the consulting room, though, for fear of faintness, it is wise to have the patient lying down upon a couch rather than sitting in a chair.

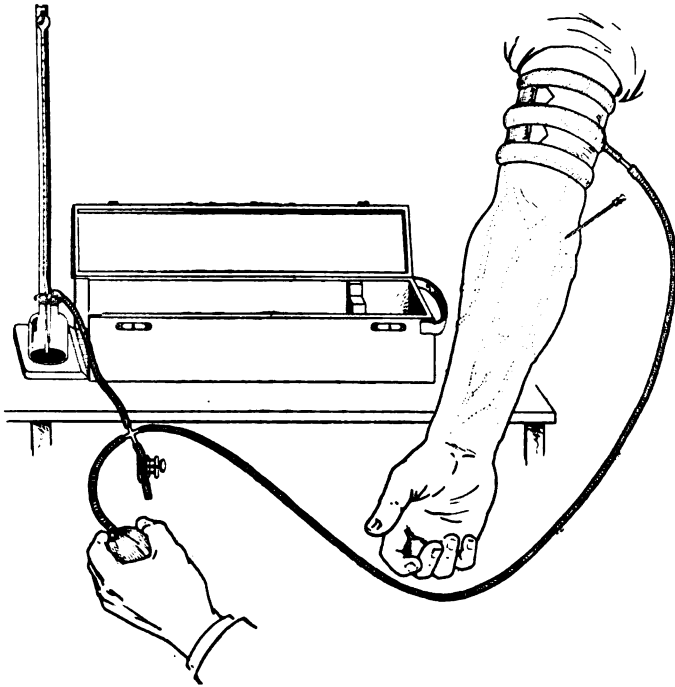


FIG. II., to illustrate the use of the sphygmomanometer armlet instead of an ordinary tourniquet in performing venesection, and the short, hollow needle through which the blood can be let out from a vein without the need for any skin incision or special subsequent dressing. The needle in the figure has not been plunged into any vein, and it should ordinarily be inserted not where it is drawn, but either into the median basilic or the median cephalic vein. It represents, however, the comparative size of the needle and more or less the direction in which the point should be at the time of insertion—namely, down the arm and not up it.

The value of the sphygmomanometer bag as a tourniquet for venesection is obvious at once: it is applied around the upper arm and the patient's arterial blood pressure can then be measured. Supposing this to be 130 mm. Hg., a little of the air can be let out of the bag until the pressure measured by the sphygmomanometer falls to 110 mm. Hg. or thereabouts. This will ensure obstruction to the return of blood through the veins whilst the arteries are still pulsating at the wrist. The flat bag is much less uncomfortable than is a narrower tourniquet, and the compressed air within it can be allowed to escape at any moment that it is desired to stop the venesection.

The accompanying diagram (Fig. II.) depicts the chief points mentioned above. The hollow needle is drawn near the arm, but it has not been inserted into a vein.

# LIST

OF

## GENTLEMEN EDUCATED AT GUY'S HOSPITAL

WHO HAVE PASSED THE

EXAMINATIONS OF THE SEVERAL UNIVERSITIES, OR OBTAINED

OTHER DISTINCTIONS, DURING THE YEAR 1911.

---

### University of London.

July.

*Examination for the Degree of Doctor of Medicine.*

Branch IV.—*Midwifery and Diseases of Women.*

W. P. H. Munden, B.S.

December.

Branch I.—*Medicine.*

C. A. Basker, B.S.

W. Johnson, B.S.

E. C. B. Ibotson, B.S.

E. L. W. Mandel, B.S.

Branch IV.—*Midwifery and Diseases of Women.*

H. C. Lucey, B.S.

W. H. Miller, B.S.

(University Medal).

*Third (M.B., B.S.) Examination for Medical Degrees.*

May.

Pass.

H. W. Catto

E. A. Penny

W. E. Fox

A. H. Todd, B.S.

G. R. Hind

H. P. Warner

October.

Honours.

E. G. Schlesinger, B.Sc. (Distinguished in Medicine).

Pass.

G. A. Blake

T. L. Jones

F. Cook, B.Sc.

J. T. M. McDougall

H. L. Hopkins

G. Marshall

D. A. Mitchell (Surgeon, R.N.)



394 *Gentlemen admitted to Degrees, &c., in the year 1911.*

Supplementary Pass List.

May.

Group I.—*Medicine.*

C. H. Crump		G. Marshall
-------------	--	-------------

Group II.—*Surgery and Midwifery.*

B. Blackwood		A. F. W. Denning
G. A. Blake		T. L. Jones
G. Covell		D. A. Mitchell
P. J. Monaghan		

October.

Group I.—*Medicine.*

V. Glendining		B. R. Parmiter
C. D. Kilpack		N. A. D. Sharp

Group II.—*Surgery and Midwifery.*

M. M. Adams		J. B. Dunning
J. H. Campain		G. F. Haycraft

*Second Examination for Medical Degrees.*

March.

Part I.

*Organic and Applied Chemistry.*

J. E. Clark		*A. S. Liebson		A. G. Simmins
H. F. T. Hogben		A. L. Punch		J. F. H. Stallman
N. E. Kendall		K. N. Purkis		J. G. Stevens
W. M. Lansdale		B. Sampson		*W. E. Tanner

\*Awarded a mark of distinction.

Part II.

*Anatomy, Physiology, and Pharmacology.*

F. D. Annesley		W. L. Gwyn-Davies		A. C. Hancock
J. R. Barrow-Clough		F. H. Dodd		T. E. Roberts
F. V. Bevan-Brown		H. W. Evans		E. D. Scott
T. P. Cole		H. Gould		W. L. Webb

July.

Part I.

*Organic and Applied Chemistry.*

C. W. W. Baxter		E. A. Levisour
A. W. A. Davies		W. H. Nicholls

Part II.

*Anatomy, Physiology, and Pharmacology.*

F. C. S. Broome		C. E. Petley
A. S. Liebson		*W. E. Tanner
H. P. Whitworth		

\*Distinguished in Pharmacology.

*First Examination for Medical Degrees.*

July.

E. S. Bowes	V. R. Hirsch	M. B. M. Tweed
O. St. L. Campion	J. G. Jones	H. S. Wachter
A. W. Cocking	F. A. Knott	L. P. Waghorn
E. N. Glover	V. E. Lloyd	M. J. T. Wallis
A. F. G. Guinness	P. G. McEvedy	K. M. C. Woodruff

December.

J. A. M. Alcock	H. M. Gray	R. L. Portway
*†L. S. Debenham	J. W. H. Grice	W. J. Vance
T. R. W. F. Figg-Hoblyn	T. L. Heath	S. Vidot

\* *Distinguished in Inorganic Chemistry.* † *Distinguished in Physics.*

*Examination for Degree of M.S.*

Branch I.—*Surgery.*

E. L. Martyn Lobb, F.R.C.S.

*Faculty of Science, B.Sc.*

*Physiology.*

A. Wills

W. A. Young

*University of Oxford.*

*Degree of D.M.*

E. L. Kennaway.

*Second Examination for M.B. and B.Ch.*

July.

R. A. Fawcus	W. H. Ogilvie	R. C. Ozanne
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December.

*Degree Conferred, B.M., B.Ch.*

T. B. Heaton.

*Second Examination for M.B., B.C.*

*Pathology.*

W. R. Reynell.

*Forensic Medicine and Public Health.*

T. B. Heaton

E. G. Martin.

*First Examination for M.B., B.Ch.*

*Organic Chemistry.*

E. W. Bowell.

**University of Cambridge.**

*Examination for the Degree of M.C.*

Easter Term.

H. Chapple

A. H. Crook

*Degree of Doctor in Medicine.*

A. S. M. Palmer.

*Degrees of M.B., B.C.*

H. L. Attwater  
A. Sandison

L. Bromley  
F. D. Saner

H. Chapple

*Third Examination for Medical and Surgical Degrees.*

Easter Term.

Part I.

*Pharmacology and General Pathology.*

G. W. B. Garrett

R. Heaton

J. M. Jarvie

Part II.

*Surgery, Midwifery and Medicine.*

H. T. Depree

A. Sandison

V. T. P. Webster

**University of Durham.**

*Degree of Doctor of Medicine for Practitioners of 15 years' standing.*

F. G. M. Philips, M.R.C.S., L.R.C.P.

*Degree of M.B.*

J. H. Owen.

*Degree of B.S.*

J. H. Owen.

*Third Examination for the Degree of Bachelor of Medicine.*

*Pathology and Elementary Bacteriology, Medical Jurisprudence and Public Health.*

Honours.—Second Class.

H. G. Dodd.

*Second Examination for the Degree of Bachelor of Medicine.*

H. H. Elliott  
D. R. Jones  
R. P. Ninnis

C. R. Smith

H. L. P. Peregrine  
P. Savage  
D. C. Scott

*First Examination for the Degree of Bachelor of Medicine.*

J. F. Carter Braine

C. C. H. Cuff

D. O. Richards

# **Royal College of Physicians of London.**

## *Examination for the Membership.*

H. B. Carlill, B.C., M.D.		E. P. Poulton, M.B., B.Ch.
E. W. Routley, M.D. (Brux.), L.R.C.P.		

## *Final Examination for the Licence.*

January.

W. T. Chaning-Pearce		N. Mutch
G. R. Hind		A. H. Todd

April.

A. M. Bodkin,	H. L. James	J. Pryce-Davies
N. S. Carruthers	W. E. Levinson	T. S. Sharpley
J. A. Delmege	A. J. McNair	H. Steinbach
H. T. Depree	H. F. Percival	H. F. Warner
L. C. D. Irvine	H. Platts	

July.

E. Billing	G. T. Foster-Smith	P. J. Monaghan
H. G. B. Blackman	V. Glendining	E. G. Schlesinger
G. A. Blake	H. L. S. Griffiths	B. T. Verver
J. H. Campain	F. G. Lloyd	M. C. Wall
H. H. Davis	G. Marshall	O. E. Williams

October.

J. B. Ball, L.D.S.	H. L. Hopkins	G. C. Lowe
B. Blackwood	Jap-ah-Chit	M. M. Munden
L. C. W. Cane	G. E. W. Lacey	B. R. Parmiter
H. Daw	W. M. Langdon	J. L. M. Symms
J. B. Hance	D. C. Lloyd	C. Worster-Drought

# **Royal College of Surgeons of England.**

## *Final Examination for the Fellowship.*

E. C. Bevers		H. I. Janmahomed, M.D.
A. H. Crook, M.C.		H. Lee
C. W. Greene		W. H. Miller
A. J. Hull, Capt., R.A.M.C.		D. Wilberforce Smith
V. Townrow		

## *Primary Examination for the Fellowship.*

H. S. Bookless		J. L. Perceval
H. Lee		H. G. Rice
H. L. Meyer		A. J. E. Smith
W. H. Trethowan		

## *Final Examination for the Membership.*

January.

W. T. Chaning-Pearce		N. Mutch
G. R. Hind		A. H. Todd

April.

A. M. Bodkin	H. L. James	J. Pryce-Davies
N. S. Carruthers	W. E. Levinson	T. S. Sharpley
J. A. Delmege	A. J. McNair	H. Steinbach
H. T. Depree	H. F. Percival	H. P. Warner
L. C. D. Irvine	H. Platts	

July.

E. Billing	G. T. Foster-Smith	P. J. Monaghan
H. G. B. Blackman	V. Glendining	E. G. Schlesinger
G. A. Blake	H. L. S. Griffiths	B. T. Verver
J. H. Campain	F. G. Lloyd	M. C. Wall
H. H. Davis	G. Marshall	O. E. Williams

October.

J. B. Ball, L.D.S.	H. L. Hopkins	G. C. Lowe
B. Blackwood	Jap-ah-Chit	M. M. Munden
L. C. W. Cane	G. E. W. Lacey	B. R. Parmiter
H. Daw	W. M. Langdon	J. L. M. Symms
J. B. Hance	D. C. Lloyd	C. Worster-Drought

*Final Examination for the Licence in Dental Surgery.*

May.

J. Benson	A. P. Marsh	G. E. H. H. Phillips
O. B. Campion	G. Matthews	G. B. Pritchard
E. J. M. Charter	H. L. Messenger	A. E. V. Spill
F. H. Edey	W. S. Ollis	O. B. Townshend
W. H. Edmonds	R. Orozco-Casorla	J. H. Wiles
G. W. E. Holloway	F. W. Paul	

October.

E. W. Bacon	L. S. Langley	I. D. Samuels
E. R. Bailey	H. L. Meyer	H. D. Shore
V. E. D. Bergh	R. Neft	T. R. Trounce
H. J. Burch	J. F. Patel	W. C. Wade
A. T. Densham	P. J. Peatfield	D. Wain
H. G. Elliott	J. G. Richards	F. R. Wallis
R. G. Farrington	R. Rodgers	C. T. Watson
W. S. Lakeman	J. H. Ross	

**Royal College of Surgeons, Edinburgh.**

*Admitted to the Fellowship.*

E. B. Hinde, M.B., B.C.	Captain C. H. Reinhold, I.M.S.
H. Jones Roberts, M.D.	

**Royal Army Medical Corps.**

D. Reynolds, M.B., B.S.

**Indian Medical Service.**

E. A. Penny, M.B., B.S. (8th place).	C. M. Plumptre (6th place)
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MEDALLISTS AND PRIZEMEN,

JULY, 1912.

*Open Scholarships in Arts.*

Thomas Albert Victor Wood, Grammar School, Wolverhampton., £100.  
Fredk. Laughton Gilbert Scott, B.A., Merton College, Oxford, £50.

*Open Scholarships in Science.*

Leonard Snowden Debenham, St. Paul's School, West Kensington, £120.  
Peter George McEvedy, Preliminary Science Class, Guy's Hospital, £60.  
James Gaymer Jones, Preliminary Science Class, Guy's Hospital, Certificate.

*Open Scholarships in Dental Mechanics.*

October, 1911, George Lambert Cutts, £20.  
May, 1912, James Millard Barnes, £20.

*Scholarship in Dental Mechanics for Pupils of Guy's Hospital.*

October, 1911, Alan Filmer Rook, £20.

*Junior Proficiency Prizes.*

William Morris Lansdale, £20.  
Arthur Lisle Punch, £15.  
Arthur Herbert Harkness, £10.  
Alfred George Simmins, Certificate.

*The Beaney Prize for Pathology.*

William Heneage Ogilvie, £34.

*The Michael Harris Prize for Anatomy.*

John Frank Herbert Stallman, £10.  
Arthur Lisle Punch, Certificate.  
Arthur Herbert Harkness, Certificate.

*The Wooldridge Memorial Prize for Physiology.*

William Morris Lansdale ) equal,  
Alfred George Simmins } £5 each.  
Arthur Lisle Punch, Certificate.  
John Stephenson, Certificate.

*The Hilton Prize for Dissections.*

1911, Humphrey Quentin Forsyth Thompson, £3.

James York Moore, £2.

1912, Martin Baird Moore Tweed, £3 10s.

Douglas Hugh Aird Galbraith. £1 10s.

*Dental Prizes.**First Year's Prize in Dental Subjects.*

William Allen Bulleid, £10.

Howell Gwyn James, Certificate.

*Second Year's Prize in Dental Subjects.*

Thomas Eric Henderson, £10.

John William Mayer, Certificate.

*Second Year's Prize in General Subjects.*

Charles Henry Housden, £10.

William Reginald Morris } Equal,  
Thomas Bernice Tustian } Certificates.

*Prize for Operative Dental Surgery.*

Frank Wentworth Lawrence, £10.

*Newland-Pedley Gold Medal for Practical Dentistry.*

Max Schneider.

*Golding-Bird Gold Medal & Scholarship in Bacteriology.*

William Leslie Webb, £20.

Richard Douglas Passey, Certificate.

*Treasurer's Gold Medal for Clinical Medicine.*

John Alfred Ryle.

*Treasurer's Gold Medal for Clinical Surgery.*

Trevor Braby Heaton } Equal.  
Harold Webb }

## THE PHYSICAL SOCIETY.

**Honorary President.**—Sir James Goodhart, Bart., M.D., LL.D.

**Honorary Vice-Presidents.**—Sir George Savage, M.D., Frederick Taylor, M.D., Charters J. Symonds, M.S.

**Presidents.**—A. H. Todd, M.B., B.S., B.Sc., G. Marshall, M.B., B.S., T. I. Bennett, H. L. James, M.B., B.S., E. G. Schlesinger, M.B., B.S. B.Sc., G. D. Eccles, R. A. Fawcus, N. Garrard, J. A. Ryle, C. S. Lane Roberts, E. D. Scott, J. L. M. Symns, T. B. Heaton, M.B., B.Ch., G. S. Miller, C. Warner, W. E. Tanner.

**Hon. Secretaries.**—G. W. Goodhart, M.A., M.D., G. T. Mullally, M.B., B.S.

## CLINICAL APPOINTMENTS HELD DURING THE YEAR 1911.

### HOUSE PHYSICIANS.

N. A. D. Sharp	G. H. Hunt	F. A. Dick
E. P. Poulton	G. Maxted	C. C. Tudge
A. S. Roe	H. W. Barber	

### HOUSE SURGEONS.

J. A. Edmond	R. Stout	F. D. Saner
G. Y. Thomson	F. Kahlenberg	T. D. M. Stout
H. Gardiner	J. G. Saner	

### ASSISTANT HOUSE SURGEONS.

F. D. Saner	G. Maxted	G. Y. Thomson
C. C. Tudge	M. R. Dobson	A. S. Roe
J. A. Edmond	R. Stout	H. T. Depree
W. E. Fox	G. T. Mullally	G. R. Hind
N. S. Carruthers	N. Mutch	A. J. McNair
	A. Sandison	

### OUT-PATIENT OFFICERS.

W. T. Clarke	H. Gardiner	E. P. Poulton
T. D. M. Stout	F. A. Dick	G. Maxted
F. D. Saner	G. Y. Thomson	C. C. Tudge
H. W. Barber	J. A. Edmond	A. S. Roe
R. Stout	H. T. Depree	G. R. Hind
	G. T. Mullally	

### OBSTETRIC RESIDENTS.

A. N. Cox	F. S. Adams	H. C. Lucey
F. Kahlenberg	W. T. Channing-Pearce	W. E. Fox



402 *Hospital Appointments held during the year 1911.*

CLINICAL ASSISTANTS.

E. Billing	M. R. Dobson	J. A. Edmond
W. E. Fox	A. S. Roe	R. Stout
H. W. Barber	H. T. Depree	G. Dunderdale
G. R. Hind	G. T. Mullally	A. Sandison
N. S. Carruthers	L. C. D. Irvine	J. M. Jarvie
A. J. McNair	N. Mutch	A. H. Todd
G. A. Blake	G. Marshall	E. G. Schlesinger
H. L. James	A. M. Bodkin	F. G. Lloyd

CLINICAL ASSISTANTS IN THE MEDICAL WARDS.

G. T. Foster-Smith	E. A. Penny	E. W. Blake
W. T. Channing-Pearce	F. G. Lloyd	N. S. Carruthers
H. H. Davis	E. R. Hart	V. Glendining
A. H. Birks	J. B. Hance	B. T. Verver
T. E. Roberts	R. Heaton	W. M. Langdon
G. B. H. Jones	E. C. Peers	H. G. Crawford
L. Milton	H. F. Stephens	T. S. Allen
S. S. Crosse	P. J. Watkin	C. Warner
W. J. D. Smyth	R. S. Bennett	W. E. Williams

CLINICAL ASSISTANTS IN THE SURGICAL WARDS.

G. Marshall	W. K. Fry	L. P. Harris
M. M. Munden	L. C. D. Irvine	H. F. Stephens
W. E. Fox	B. R. Parmiter	F. C. Newman
J. B. Hance	G. W. M. Andrew	C. M. Ryley
H. G. Crawford	A. L. Gardner	W. M. Langdon
	H. C. Godding	

CLINICAL ASSISTANTS IN THE MEDICAL OUT-PATIENTS.

A. J. Fradersdorff	G. E. W. Lacy	R. G. Oram
A. C. Jepson	J. L. M. Symns	W. S. George

SURGEONS' DRESSERS.

W. S. George	C. Warner	V. F. Soothill
J. H. Joly	C. Worster-Drought	J. P. Jones
R. S. Kennedy	F. C. Hunôt	F. C. Newman
E. L. Jones	H. C. Godding	O. R. L. Wilson
H. J. Hoby	L. B. Stringer	S. Wickenden
A. Tilbury	M. Scott	R. D. Passey
T. I. Bennett	J. L. D. Lewis	L. M. Menage
A. L. Shearwood	H. Webb	K. B. Clarke
G. W. King	W. Matthews	W. K. Fry
W. C. Whitworth	L. P. Harris	A. Samuel
E. C. Cline	J. F. G. Richards	H. C. Rook
C. H. G. Pochin	S. Keith	A. G. H. Moore
G. S. Miller	A. B. Danby	W. Robinson
G. A. Pratt	G. E. Genge-Andrews	W. C. D. Maile
F. B. Bull	W. H. Ogilvie	W. R. Reynell
J. A. Martin	A. M. Zamora	W. L. Webb
H. Sharpe	E. M. Mahon	G. D. Eccles
F. Collar	A. C. Clifford	D. V. Pickering
C. Sherris	K. J. T. Keer	O. G. Morgan
J. L. Perceval	F. A. Hampton	D. W. John
R. A. Fawcus	R. C. Ozanne	N. Garrard
E. G. Martin	J. A. Ryle	R. Creasy

OPHTHALMIC HOUSE-SURGEONS.

H. L. Attwater

V. Glendining

OPHTHALMIC DRESSERS.

N. Mutch	T. B. Heaton	A. J. McNair
H. P. Warner	A. L. Gardner	H. L. James
R. C. Poyser	H. L. S. Griffiths	E. G. Schlesinger
G. Marshall	G. Covell	G. A. Blake
A. S. Seabrooke	A. C. Jepson	F. G. Lloyd
B. Blackwood	F. Cook	H. L. Hopkins
C. Aldis	V. F. Soothill	F. S. Adams
J. L. M. Symms	W. E. S. Digby	E. W. Blake
E. R. Hart	T. S. Allen	E. C. Peers
P. Smith	T. P. Cole	D. A. Davies
M. Pern	C. A. R. Gatley	

DRESSERS IN THE THROAT DEPARTMENT.

W. P. Vicary	G. R. Hind	G. T. Mullally
F. Cook	A. C. Jepson	A. S. Seabrooke
T. S. Allen	G. T. Foster-Smith	F. G. Lloyd
E. A. Penny	A. H. Todd	O. E. Williams
B. Blackwood	W. E. S. Digby	H. P. Warner
H. L. Hopkins	H. L. James	E. G. Schlesinger
C. Aldis	T. P. Cole	C. A. R. Gatley
A. H. Birks	A. J. McNair	T. B. Heaton

ASSISTANT SURGEONS' DRESSERS.

G. A. Pratt	A. V. Moberly	L. M. J. Menage
H. Sharpe	H. G. Pochin	G. S. Miller
J. F. Richards	A. G. H. Moore	S. Keith
R. Creasy	D. W. Jones	A. S. Erulkar
G. Genge-Andrews	T. A. Townsend	H. C. Rook
J. L. Perceval	W. Robinson	A. S. Danby
F. B. Bull	W. C. D. Maile	E. H. Mahon
N. Garrard	V. Atienza	K. J. Keer
A. C. Clifford	R. C. Ozanne	D. V. Pickering
O. G. Morgan	C. Sherris	H. Sharpe
E. G. Martin	D. W. John	A. M. Zamora
F. Collar	W. H. Ogilvie	F. A. Hampton
R. H. Lucas	G. D. Eccles	J. L. Lauder
R. P. Ninnis	M. Z. Hanafy	F. L. Spalding
H. W. Evans	A. J. Drew	A. K. Selim
E. S. Taylor	H. Gould	E. D. Scott
R. C. Matson	P. H. Berry	C. H. L. Harper
W. R. Pryn	J. R. Barrow-Clough	J. W. Kemp
F. L. Lawson	L. D. Wright	A. M. Henry
W. L. Gwyn-Davies	A. C. Hancock	H. Mather
D. C. Scott	H. S. Groves	C. H. G. Penny
A. J. E. Smith	F. D. Anuesley	A. Abd-el-Al

DENTAL SURGEONS' DRESSERS.

A. J. McNair	O. E. Williams	H. H. Davis
G. A. Blake	H. L. James	E. C. Peers
H. P. Warner	G. C. Lowe	D. A. Davies
S. S. Crosse	W. W. Cook	W. E. S. Digby

CLERKS IN THE SKIN DEPARTMENT.

N. S. Carruthers	O. R. L. Wilson	G. E. Genge-Andrews
J. L. Perceval	R. A. Fawcus	J. A. Ryle
E. S. Taylor	B. R. Parmiter	

# AURAL SURGEONS' DRESSERS.

H. G. Crawford	O. E. Williams	R. Heaton
J. M. Jarvie	A. H. Todd	F. G. Lloyd
T. S. Allen	N. Mutch	E. W. Blake
E. R. Hart	J. L. M. Symns	H. L. Hopkins
W. M. Langdon	D. A. Davies	M. Pern

# MEDICAL WARD CLERKS.

A. L. Shearwood	W. K. Fry	T. I. Bennett
R. D. Passey	W. Matthews	G. W. King
K. B. Clarke	W. C. Whitworth	E. C. Cline
L. P. Harris	L. B. Stringer	O. R. L. Wilson
H. J. Hobby	S. Wickenden	A. Tilbury
M. Scott	J. L. D. Lewis	H. Webb
J. A. Ryle	C. Sherris	F. Collar
A. M. Zamora	A. C. Clifford	R. A. Fawcus
N. Garrard	F. A. Hampton	D. W. John
E. G. Martin	J. A. Martin	O. G. Morgan
W. H. Ogilvie	R. C. Ozanne	D. V. Pickering
W. R. Reynell	G. D. Eccles	J. F. G. Richards
H. C. Rook	A. B. Danby	G. A. Pratt
W. C. D. Maile	S. Keith	C. H. G. Pochin
G. S. Miller	J. L. Perceval	G. E. Genge-Andrews
A. G. H. Moore	W. Robinson	D. R. Jones
A. M. Henry	A. S. Erulkar	V. Atienza
R. Creasy	F. B. Bull	M. Z. Hanafy
E. M. Mahon	H. S. Groves	R. H. Lucas
A. Abd-el-Al	K. J. T. Keer	C. H. G. Penny
L. D. Wright	J. W. Kemp	A. K. Selim
W. R. Pryn	F. L. Spalding	C. H. L. Harper
T. A. Townsend	E. S. Taylor	P. H. Berry
H. Sharpe	F. D. Annesley	H. Mather
W. L. Gwyn-Davies	A. C. Hancock	A. V. Moberly
D. C. Scott	A. J. E. Smith	R. O. H. Jones
F. H. Dodd	C. S. L. Roberts	A. H. Taymour
P. Savage	J. York Moore	J. R. Barrow-Clough
A. J. Drew	H. W. Evans	H. Gould
W. K. McKay	C. G. McClymont	R. C. Matson
R. P. Ninnis	E. D. Scott	L. Du Vergé
P. R. Chevreau	F. V. Bevan-Brown	F. W. Lawson
C. Lambrinudi	J. F. Venables	C. F. Pedley

# SURGICAL WARD CLERKS.

R. H. Lucas	C. H. G. Penny	E. S. Taylor
M. Z. Hanafy	C. H. L. Harper	P. H. Berry
A. K. Selim	A. Abd-el-Al	J. W. Kemp
W. R. Pryn	F. L. Spalding	A. M. Henry
H. S. Groves	L. D. Wright	F. D. Annesley
D. C. Scott	R. P. Ninnis	E. D. Scott
J. R. Barrow-Clough	W. L. Gwyn-Davies	A. V. Moberly
A. J. Drew	R. J. Matson	F. W. Lawson
A. C. Hancock	H. Mather	H. Gould
R. O. H. Jones	F. H. Dodd	C. S. L. Roberts
W. K. McKay	C. Lambrinudi	C. F. Pedley
J. F. Venables	A. H. Taymour	J. York Moore
F. V. Bevan-Brown	T. W. Sheldon	C. G. McClymont
P. R. Chevreau	P. Savage	W. E. Tanner
S. Wilson	F. C. S. Broome	D. R. Jones
H. P. Whitworth	H. H. Elliott	C. Dean
W. D. Galloway	H. L. P. Peregrine	E. C. W. Starling
L. M. Smith	A. N. Minns	D. McManus
P. R. Boswell	R. J. Hearn	

POST-MORTEM CLERKS.

W. R. Reynell	R. C. Ozanne	H. L. James
W. H. Ogilvie	T. E. Roberts	F. A. Hampton
R. A. Fawcus	V. Glendining	T. A. Townsend
C. H. Harper	S. S. Crosse	H. N. Eccles
W. S. George	J. L. Stewart	E. Lawrence Jones
E. W. Blake	F. C. Hunôt	L. Milton
E. R. Hart	V. A. Luna	G. D. Eccles
C. M. Ryley	W. M. Langdon	R. D. Passey
H. J. Hoby	A. L. Shearwood	M. Scott
S. Wickenden	O. R. L. Wilson	W. Matthews
H. Webb	W. J. D. Smyth	C. Warner
H. C. Godding	F. C. Cline	A. G. H. Moore
J. M. Joly	C. H. G. Pochin	G. S. Miller
W. Robinson	J. F. G. Richards	

OBSTETRIC DRESSERS.

J. Pryce Davies	H. L. Hopkins	H. L. Griffiths
E. G. Reeve	P. Smith	E. G. Schlesinger
T. S. Allen	A. Sandison	J. L. M. Symns
G. Covell	V. Glendining	D. A. Davies
W. J. D. Smyth	G. W. B. Garrett	T. B. Heaton
M. Pern	V. A. Luna	F. Cook
C. Aldis	T. P. Cole	C. A. R. Gatley
E. C. Peers	H. C. Godding	J. L. Stewart
J. M. Joly	E. L. Jones	F. C. Hunôt
W. S. George	R. S. Kennedy	C. Warner
L. B. Stringer	M. Scott	A. Tilbury
H. J. Hoby	T. A. Townsend	O. R. L. Wilson
T. I. Bennett	R. D. Passey	A. L. Shearwood
H. V. Leigh	S. Wickenden	K. B. Clarke

EXTEEN OBSTETRIC ATTENDANTS.

E. W. Blake	H. P. Warner	G. T. Foster Smith
F. G. Lloyd	G. Marshall	R. C. Poyser
L. Milton	T. E. Roberts	P. J. Watkin
A. H. Birks	H. N. Eccles	G. B. H. Jones
B. R. Parmiter	C. F. Constant	G. W. M. Andrew
P. Smith	J. L. Stewart	G. Covell
E. G. Schlesinger	H. L. Hopkins	H. G. Crawford
J. L. M. Symns	W. E. S. Digby	S. S. Crosse
C. Warner	C. Worster-Drought	F. Tooth
V. A. Luna	C. Aldis	F. Cook
T. P. Cole	D. A. Davies	G. W. B. Garrett
T. B. Heaton	C. A. R. Gatley	W. J. D. Smyth
E. C. Cline	W. C. Whitworth	M. Pern
J. L. D. Lewis	L. B. Stringer	J. M. Joly
W. S. George	R. S. Kennedy	H. C. Godding
F. C. Hunôt	W. Matthews	G. E. Genge-Andrews
H. Webb	E. L. Jones	S. Keith
F. C. Newman	V. F. Soothill	J. P. Jones
V. Atienza	A. S. Erulkar	

CLERKS IN THE ELECTRICAL DEPARTMENT.

C. Aldis	C. A. R. Gatley	C. E. Reckitt
T. I. Bennett	G. W. B. Garrett	V. F. Soothill
V. A. Luna	C. Warner	C. Worster-Drought

CLERKS IN OUT-PATIENTS FOR NERVOUS DISEASES

H. L. Hopkins	E. R. Hart	J. H. Campaign
G. A. Blake	S. Wickenden	C. Worster-Drought
S. S. Crosse	B. R. Parmiter	J. L. M. Symns

# DENTAL APPOINTMENTS HELD DURING THE YEAR 1911.

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## DENTAL HOUSE SURGEONS.

W. H. Wotton	F. S. Glover	G. W. E. Holloway
G. B. Pritchard	H. L. Messenger	(3 months)
R. B. Campion (3 months)	H. F. Barge	

## ASSISTANT DENTAL HOUSE SURGEONS.

R. B. Campion	J. H. Rhodes	W. S. Lakeman
H. L. Messenger	G. B. Pritchard	I. G. Samuels
R. R. Adams	E. R. Bailey	F. J. Tipper
E. N. Bacon	H. D. Shore	D. Wain

## ASSISTANT DEMONSTRATORS IN DENTAL MECHANICS.

R. G. Farrington	A. J. Percy	L. S. Langley
	W. S. Stranack	

## ASSISTANT DEMONSTRATORS IN DENTAL METALLURGY.

H. B. Neely	H. L. Messenger	D. Wain
	T. E. Henderson	

## ASSISTANT DEMONSTRATORS OF DENTAL MICROSCOPY.

R. D. Tanner	E. W. Bacon
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## DRESSERS IN THE GAS ROOM.

C. A. Pollard	R. D. Tanner	E. W. Bacon
H. G. Elliott	A. F. Camp	C. A. Achner
C. T. Watson	H. C. Corke	I. G. Samuels
S. J. F. Webb	V. E. D. Bergh	G. Matthews
A. J. Percy	E. L. Fraser	J. F. Patel
R. Orozco-Casorla	L. S. Langley	W. J. O'Kane
W. S. Ollis	C. J. M. Charter	J. S. Cocks
R. Rodgers	E. R. Bailey	T. R. Trounce
S. W. Bevis	P. J. Peatfield	A. T. Densham
C. H. Stainer	H. N. Purdom	E. S. Hockett
F. Wisher	D. Wain	C. D. Neale
J. H. Ross	F. R. Wallis	H. L. Meyer
T. E. Henderson	C. Wade	J. H. H. Griffin
P. J. Peatfield	J. E. R. Evans	G. H. Warner
J. G. Richards	H. J. Burch	A. J. Chapman
T. B. Tustian	H. D. Shore	R. G. Farrington
C. H. Housden	W. R. Morris	S. W. Bevis
J. S. Cocks	W. S. Stranack	R. M. Veale
C. M. Desai	E. R. Williams	T. F. H. Gollledge
I. W. Pasmore	D. H. Barr	

**DRESSERS IN THE EXTRACTION ROOM.**

T. E. Henderson	I. W. Pasmore	W. H. Glover
R. W. Spong	F. W. Lawrence	W. Adderley
J. S. Palmer	A. D. Buck	M. F. Hopson
D. B. Morrish	J. W. Mayer	S. Hanreck
P. S. McFarlane	L. T. Montgomery	R. W. Ballard
A. S. Clarke	H. L. Meyer	J. S. Cocks
R. L. Donn	A. G. Forrest	W. H. C. Gordon
O. N. Mash	E. C. Nicholls	R. H. Gaverick
C. Glover	G. K. Moore	T. P. Cooper
R. Boutwood	H. B. Neely	L. D. Windermere
A. D. Buck	F. J. Neale	F. H. Morrell
S. Adams	G. F. Charles	J. W. Mayer
C. E. Thomas	S. Stevens	E. E. Johnson
E. C. Rycroft	A. J. Barber	R. H. Rix
L. C. Cohen	H. C. Duggan	C. F. Haine
B. Isaacs	E. R. Saul	R. C. W. Staley
A. H. Cole	P. King	J. L. Garrard
H. G. James	W. A. Easton	W. G. S. Neely.

**CASUALTY DRESSERS.**

E. R. Williams	P. J. Peatfield	J. H. Ross
W. S. Stranack	C. J. Phillips	W. J. O'Kane
R. M. Veale	R. R. Steele	D. H. Barr
W. R. Morris	A. T. Densham	C. H. Housden
M. Schneider	E. R. Bailey	J. S. Sutton
T. E. Henderson	V. T. H. Gollledge	T. B. Tustian
H. L. Meyer	J. H. H. Griffin	I. W. Pasmore
W. T. Flooks	G. L. Pemberton	W. Adderley
A. S. Clarke	R. W. Spong	W. H. Glover
A. D. Buck	F. W. Lawrence	D. B. Morrish
W. L. Partridge	J. W. Mayer	H. B. Neely
J. S. Palmer	A. G. Forrest	J. E. Davies
L. R. Pickett	O. N. Mash	C. H. Medlock
T. P. Cooper	P. S. McFarlane	G. K. Moore

# GUY'S HOSPITAL.

## MEDICAL AND SURGICAL STAFF.

1912.

**Consulting Physicians.**—P. H. PYE-SMITH, M.D., F.R.S.; SIR JAMES GOODHART, BART., M.D., LL.D.; F. TAYLOR, M.D.

**Consulting Surgeons.**—THOMAS BRYANT, M.Ch.; SIR H. G. HOWSE, M.S.; W. H. A. JACOBSON, M.Ch.; R. CLEMENT LUCAS, B.S.; C. H. GOLDING-BIRD, M.B.; CHARTERS J. SYMONDS, M.S.

**Consulting Obstetric Physician.**—A. L. GALABIN, M.D.

**Consulting Physician for Mental Diseases.**—SIR GEORGE SAVAGE, M.D.

**Consulting Ophthalmic Surgeons.**—C. HIGGINS, Esq.; W. A. BRAILEY, M.D.

**Consulting Aural Surgeon.**—W. LAIDLAW PURVES, M.D.

**Consulting Dental Surgeons.**—F. NEWLAND-PEDLEY, Esq.; W. A. MAGGS, Esq.

**Consulting Anæsthetist.**—TOM BIRD, Esq.

### Physicians and Assistant Physicians.

W. HALE WHITE, M.D.  
G. NEWTON PITT, M.D.  
SIR E. COOPER PERRY, M.D.  
L. E. SHAW, M.D.

J. FAWCETT, M.D.  
A. P. BEDDARD, M.D.  
H. S. FRENCH, M.D.  
A. F. HERTZ, M.D.

H. C. CAMERON, M.D.

### Surgeons and Assistant Surgeons.

W. ARBUTHNOT LANE, M.S.  
L. A. DUNN, M.S.  
SIR ALFRED FRIPP, M.S., C.B.,  
K.C.V.O.  
F. J. STEWARD, M.S.

C. H. FAGGE, M.S.  
R. P. ROWLANDS, M.S.  
P. TURNER, M.S.  
E. C. HUGHES, M.C.  
R. DAVIES-COLLEY, M.C.

### Obstetric Surgeons.

J. H. TARGETT, M.S.

G. BELLINGHAM SMITH, M.B., B.S.

### Ophthalmic Surgeons.

H. L. EASON, M.S., M.D.

A. W. ORMOND, Esq.

### Surgeons in Charge of Throat and Ear Department.

W. M. MOLLISON, M.C.

T. B. LAYTON, M.S.

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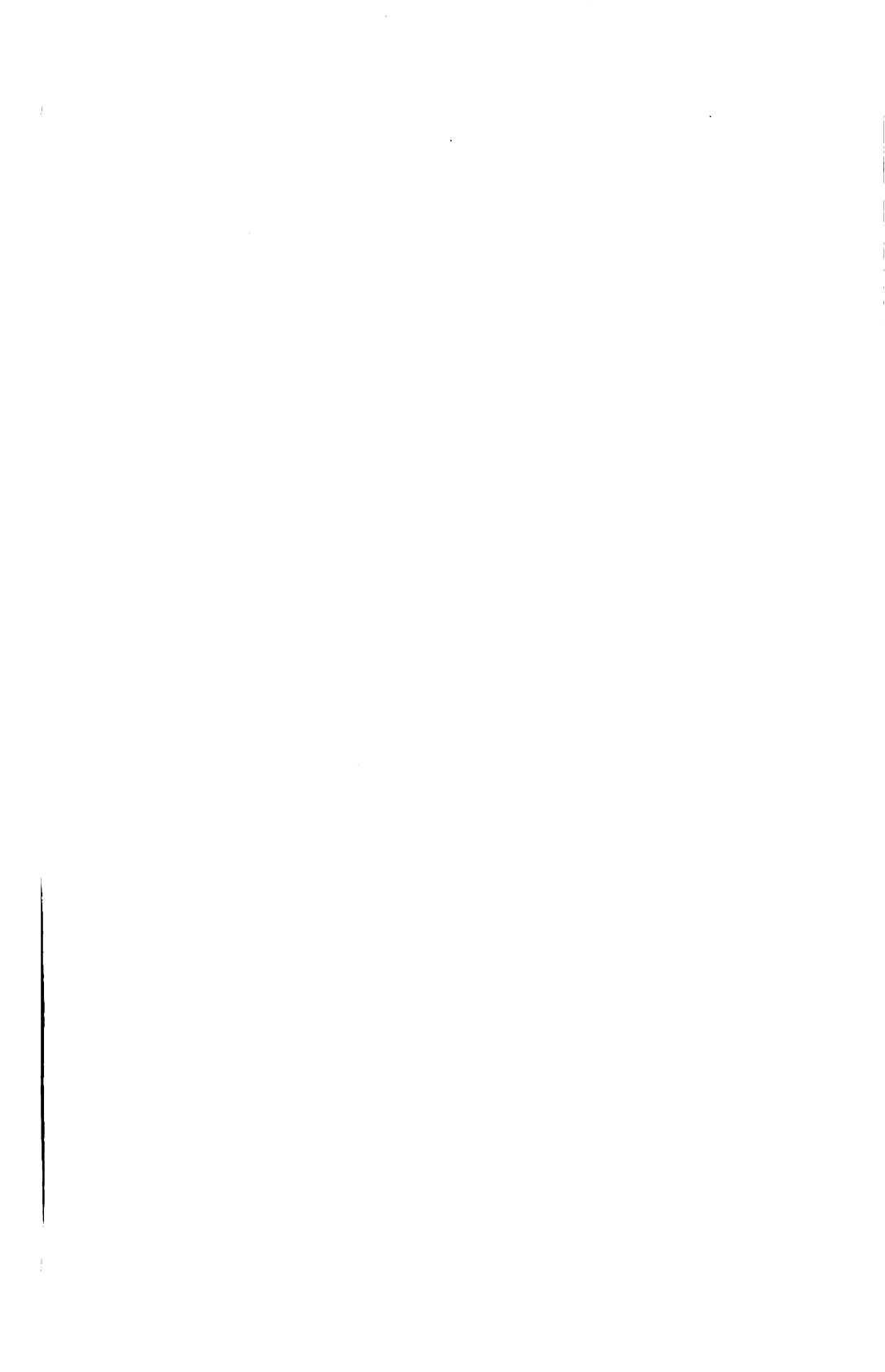
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The Physical Society, 1910—11.  
Clinical Appointments held during the year 1910.  
Dental Appointments held during the year 1910.  
List of Original Papers by Members of Guy's Staff contributed to the Medical Press during the year.  
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